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MEMBER OF THE OPHTHALMIC STAFF.

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PREFACE.

THE first volume of the PHILADELPHIA HOSPITAL REPORTS was published in 1890 and the second in 1893. It was hoped that the present volume would appear in 1895, but unavoidable delays have prevented its publication until the present time. Historical and biographical matters have been almost entirely omitted, and the bulk of the volume is composed of clinical contributions by the members of the Staff, aided in some instances by the Resident Physicians. With the exception of the first medical paper and one historical note, these contributions are largely the record and study of cases occurring in the hospital itself.

The various departments of the hospital are represented as follows: Seven papers from the Medical, three from the Surgical, three from the Obstetrical and Gynecological, four from the Neurological, together with ten reports of cases occurring in the services of various members of the Neurological Staff, recorded by the Resident Physicians, one from the Ophthalmic, one from the Ophthalmic and Neurological combined, two from the Dermatological, and two from the Pathological Department—in all, twenty-nine scientific communications.

The frontispiece represents the Nurses' House of the Philadelphia Hospital Training School—one of the most notable recent additions to the hospital. Other improvements are described in the article kindly contributed by Mr. Charles Lawrence, Superintendent of the Philadelphia Almshouse and Hospital. Acknowledgments are due to Dr. E. R. Stone and Dr. W. A. N. Dorland for the list of ex-Resident Physicians of the Philadelphia Hospital, and to Dr. Charles K. Mills, the editor of the two previous volumes, for many valuable suggestions.

G. E. DE SCHWEINITZ, M.D.

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ILLUSTRATIONS.

RECENT ADDITIONS AND IMPROVEMENTS IN THE PHILADELPHIA HOSPITAL.

By CHARLES LAWRENCE, Superintendent.

Since the publication of Volume II. of the "Philadelphia Hospital Reports," numerous additions and improvements have been made in various departments of the institution. The following may be mentioned as some of the most important:

All of the wards, corridors and stairways in the entire institution have been stripped of the old plaster, replastered with adamantine cement and painted; all the old flooring, joists and other woodwork that were decayed, were removed and replaced by new. Partition walls were torn out and arches built, making large, airy wards out of narrow ones; the last remnants of the old cubbies have been removed; in fact it may be said that little remains of the old buildings but the walls and roofs.

The improvements made in the Insane Department were so manifest, that Hon. Mahlon H. Dickinson, President of the State Board of Charities, remarked, when visiting one of the wards, "I do not believe that there is an institution in the world that has a ward equal to this." Cadwalader Biddle, Esq., Secretary and Agent of the same Board, upon the occasion of a visit to wards 6, 7 and 8, after they had been remodelled, said, "Nothing short of an inspiration suggested such a great improvement as this."

The stone wall around the building has been completed, making about a mile in length. A brick carriage and ambulance house, 121 x 20 feet, has been erected to take the place of the old wagon sheds. The stable grounds are well lighted by electricity, and now there are three ambulances in constant service.

More than 60,000 square feet of cement walks have been laid, and the grounds beautified by flower beds in a number of places. A

variety of plants, such as geraniums, cannas, begonias, roses, fuchsias, coleus, ferns, heliotropes, calendulas, verbenas, petunias, etc., were planted, making a very creditable display. A garden has been laid out in the courtyard, on the male side; a fountain plays in the center of it. In order to have a full supply of flowers, a conservatory 70 x 20 feet was erected, where propagating beds furnish the plants. The whole is known as "Lawrence Park," it having been so named by the Bureau of Charities.

The erection of a building for the nurses of the Training School of the Hospital will effect a great change in the accommodations. The structure is 134 feet 8 inches long and 78 feet 10 inches wide, with corridors 10 feet wide the entire length of each story, terminating in bays, with transverse corridors, 7 feet wide, also terminating in bays, and with broad stairways at the intersections of corridors at each end of the building. The first story is 12 feet high in the clear, the second and third stories each 9 feet high, plastered on hollow brick walls with hard polished finish coat cement, washboards or bases, and rounded corners throughout, the amount of wood finish being reduced to a minimum.

There are total accomodations on the three main floors for 120 nurses, exclusive of the fourth story which will have a central dormitory about 62 x 34 feet and two (north and south) attics 25 x 60 feet each. The exterior is of simple hard brick with marble and terra cotta trimmings, the roofing is of green slate with copper flushings and gutters, and the porches on the east and west fronts are supported by brick arches and terra cotta with marble coping, the asphalt roofs of the porches serving for an open balcony, heavy glass floor lights having been introduced to prevent darkening of lower rooms.. (See Frontispiece.)

The wards and rooms in the Hospital building that were occupied by the nurses have been remodelled, replastered and painted. On the lower floor, the rooms formerly occupied as an office and a dining-room for the Chief Nurse and assistant, have been converted into a receiving ward with bathrooms, etc. The reception-room adjoining is now a ward for emergency cases, and the Drug Department has been enlarged by the addition of what was formerly a class-room.

These changes are a great improvement on the old receiving ward building across the street. The patients will be brought directly to the Hospital, and need not be exposed to the air after the bath, as

was the case under former conditions. The additional wards will afford accommodations for about 150 patients, thus increasing the capacity to more than 1300 beds.

During the year 1894, there were 7930 cases treated in the Hospital; 28 less than in 1893. There were 1529 patients treated in the Insane Department during the year 1894. The average daily population of the institution for the year was 3432, and during the month of December it was 3513.

On the first of January, 1894, the inmates numbered	3,637
Admitted during the year	6,412
Born " "	112
Total	10,161
Discharged	4,955
Overstayed liberty	569
Died	1,054
Total	6,578
Remaining, December 31, 1894	3,583

APPENDICITIS.

By JAMES TYSON, M.D.

The word appendicitis, which is now by almost unanimous consent applied to the disease under consideration, did not secure the application without a struggle. The term typhlitis, so long employed, was adopted because it was thought that the disease began in the cæcum. Modern studies go to show with almost absolute certainity that it never begins in the cæcum, but that the appendix in all cases is the root of the evil. Indeed it is extremely doubtful whether there is such a disease as typhilitis or cæcitis in the strict sense of the term. It does often happen that the earliest symptoms by which appendicitis is recognized are those of inflammation of the peritoneum, covering the appendix and adjacent cæcum, but the existence of very positive disease of the mucous membrane of the appendix has been demonstrated over and over again where the peritoneum has not been invaded. It is, therefore, likely that the process begins in the appendicular mucous membrane each time. The term appendicular peritonitis is a good one for the inflammation of the peritoneal covering of the appendix, while perityphlitis is equally suitable for the more extensive peritonitis about the cæcum, and the term perityphilitic abscess indiates well that the same inflammation has gone on to pus formation.

History.—None of the facts bearing on the nature of appendicitis are of very old date, while the correct notion of its nature may be said to be quite recently established. The first recorded case of perforation of the vermiform appendix appears to have been by Mestivier, in 1759, caused by a large pin in the appendix; another was reported by J. Parkinson, an English physician, in 1812; another by Wegeler, in 1813. Other isolated cases of fatal inflammation of the appendix were published from time to time, but the first systematic article was prepared by Husson and Dance, in 1827, at the suggestion of Dupuytren, and the views promulgated by them were apparently those of the

great surgeon himself, since they are the same as those he published six years later in his "Lectures on Clinical Surgery." He treats of irritation and inflammation of the mucous membranes of the cæcum, extending thence to the retro-cæcal tissue and thence rarely to the peritoneum. The appendix is totally ignored. In 1830, Goldbeck, at the suggestion of Puchelt, of Heidelberg, wrote his graduation thesis, "On a Peculiar Inflammatory Tumor of the Right Iliac Region." He adopted the views of the French authors and called the disease perityphlitis. He, also, recorded a case of perforation of the appendix with resulting peritonitis. He says, moreover, that in fatal cases of perityphlitis, the appendix has been found intact.

In 1831, J. M. Ferrall published a paper, said to have been written several years earlier, on "Phlegmonous Tumors in the Right Iliac Region," in which the cæcum is also held to be the primary seat of the phlegmon, which is described as extending thence to the connective tissue behind it, the peritoneum being accorded a minor *rôle*.

In 1834, James Copland, in his "Dictionary of Practical Medicine," describes what we now know as perityphlitis, and distinguished between it and inflammation of the cæcum. He, moreover, recognized the appendix as a possible primary seat of disease in this region, due to foreign bodies in it and terminating in gangrene—a great advance over anything before him. John Burne came still nearer the truth in 1837, and again in 1839. Though he wrote on "Inflammation of the Cæcum," even in his first paper he speaks of "ulceration of the appendix," set up by foreign bodies, such as raisin seeds, cherry stones and concretions, of possible perforation resulting in general peritonitis or local peritonitis, with abscess. In his second paper he goes farther and states his belief that all Dupuytren's cases were due to disease of the appendix. He introduces the term *tuphlo-enteritis*.

In 1838, J. F. H. Albers retrograded a little. Publishing a paper on inflammation of the cæcum, and introducing the term *typhlitis*, which he divides into acute, chronic and stercoral typhlitis with perityphlitis, he distinguishes the latter affection from typhlitis, with which he says Puchelt and others confounded it. But while recognizing the possibility of disease starting in the appendix and going on even to perforation, he regarded it as an event secondary to disease of the cæcum. In the next year Grisolle, appreciating correctly the *rôle* played by perforation of the appendix in causing the iliac phlegmon and abscess,

opposed the teaching of Albers and claimed that the cæcum could not cause the grave effects ascribed to it, since dysenteric and other well recognized forms of ulceration of the same structure show no tendency to extend into the neighboring connective tissue. Grisolle, as though still under the thraldom of Dupuytren and the French school, assigned an important *rôle* to the cæcum.

From this time, however, and indeed from the data of Burne's paper in 1837 to the present, appendicitis has been an acknowledged disease, but it has seemed almost impossible, even up to this day, to shake off the idea of a typhlitis as a responsible factor in the phenomena of appendicitis. Villermey, in 1840, reported some cases of rapidly fatal inflammation and gangrene of the appendix. In 1843, A. Voltz published a retrospective paper entitled "Ulceration and Perforation of the Appendix," occasioned by foreign bodies. He concluded that the appendix was the organ at fault in all cases previously published, and apparently for the first time the cæcum and retrocæcal tissues are ignored.

Simple catarrh of the appendix was first recognized by Rokitansky, in 1843, in his classic work on "Pathological Anatomy." He ascribes it to the irritation of fæcal matter and to concretions, and contrasted it with the more intense processes of gangrene and perforation. Such inflammation, he says, may become chronic or go on to ulceration. He also refers to the benign effect of inflammatory adhesions in protecting against general peritonitis, in the event of subsequent perforation. He still admits the existence of catarrhal inflammation of the cæcum, ulceration and perforation of the latter, with inflammation of the postcæcal tissue as a consequence. So, G. Lewis, in 1856, ascribed the less serious consequences, including, however, suppuration, to typhlitis, while the violent and fatal cases, he said, began with appendicitis, induced always by concretions. In 1858, C. Wister attached further importance to the part of the appendix in producing the symptoms in question. In this year, too, Oppolzer suggested the name *paratyphlitis* for that form of iliac phlegmon which was extraperitoneal; *i. e.*, between the iliac fascia and bone.

Samuel Wilks was one of those who appreciated the *rôle* of the appendix. Thus, in the treatise of Wilks and Moxon on "Pathological Anatomy," in 1875, he says, referring to the terms cæcitis, typhlitis and perityphlitis: "It is not clear, however, that any one particular form of

disease is intended by those who make use of these expressions. The cases to which these names are given frequently occur clinically and recover; but when disease in the same region with similar characters proves fatal, we find usually some prior morbid process in the appendix rather than in the cæcum itself." Also, "the suddenness of the attack of cæcitis, and the local peritonitis following, even in the large number of cases which recover, all point to the appendix as being the most frequent cause." But he says, also: "Inflammations of the cæcum itself do occur, and apparently are sometimes caused by the continuous lodgment of hard fæces in this part of the intestines. Such inflammations, by ulcerating the mucous membrane, lead to perforation and local peritonitis, forming fæcal abscess which may discharge inwards, but we believe that this is comparatively rare." Dr. Wilks's most recent views are perhaps best expressed by C. Hilton Fagge, who, in his "Practice of Medicine," edition of 1886, says: "Dr. Wilks has repeatedly expressed to me the opinion that, in both 'typhlitis and perityphlitis,' the disease begins in the appendix, and that variations in the intensity of the morbid process are the real cause of the supposed distinction between them. And, so far as I can learn, all the evidence which morbid anatomy affords points strongly in that direction." C. With, of Copenhagen, was, however, apparently the first to deny pointedly, in 1880, that peritonitis ever originate in typhlitis. Even Reginald H. Fitz, in his epoch-making paper, read before the Association of American Physicians in 1886, admitted, as an extreme rarity, a primary perforating inflammation of the cæcum with which appendicitis may be confounded.

The text-books, published prior to 1892, very generally treat of typhlitis as an important factor in producing the ultimate phenomena of what is now known as appendicitis, unless we except that of Dr. Fagge, already quoted, who, while he uses the word typhlitis, evidently means by it disease of the appendix. Ziegler, in his "Pathological Anatomy" (1885), also uses the term typhlitis for appendicitis. William Osler, in his edition of 1892, says that the terms "perityphlitis and paratyphlitis should be altogether discarded, as the cases are, with rare exceptions, due to disease of the vermiform appendix," and says, also, of "typhlitis or inflammation of the cæcum, that it is a doubtful and uncertain malady, the pathology of which is not known, but which, clinically, is still recognized by authors."

Three special treatises of great value have recently been published. "The Pathology of the Vermiform Appendix," by T. N. Kelynack, of Manchester, England, in 1893; "Appendicitis," by George R. Fowler, of New York, in 1894; and "Diseases of the Vermiform Appendix," by Herbert P. Hawkins, of London, in 1895. The latter summarizes the situation in the following proposition, to the confirmation of which American surgery has largely contributed in the large experience of operators like Maurice H. Richardson, of Boston; Charles McBurney and Robert F. Weir, of New York; John B. Deaver, of Philadelphia, and N. Senn and J. B. Murphy, of Chicago:—"in fact, it will be generally allowed that a perforating ulcer of the cæcum, though it does certainly occur, is of so rare occurrence that it may be disregarded;" also—"there is ample evidence that appendicular disease is, at any rate, of frequent occurrence; and this frequency, moreover, is sufficiently frequent to justify us in regarding the appendix as the sole cause of all cases of perityphlitis, mild or severe." In a historical sketch mention should not be omitted of Willard Parker as the first to suggest operation.

Pathology and Morbid Anatomy.—The etiology of appendicitis will be more easily understood if its morbid anatomy is first considered. Modern studies establish the existence of three degrees of appendicitis: I. Catarrhal appendicitis; II. Ulcerative appendicitis; III. Infective appendicitis.

I. *Catarrhal Appendicitis.*—Our knowledge of this is based upon the systematic minute study of cases which come to autopsy from other causes, as well as from operation. They are apparently independent of the presence of fæcal matter or foreign bodies. In the acute form there is a shedding of the epithelium of the mucous membrane with detachment, partial destruction and extrusion of the follicles of Lieberkühn, with some cellular infiltration of the retiform tissue at their base. The lumen of the appendix contains mucus, leucocytes, exfoliated cells, and casts more or less perfect, of the crypts, with granular débris from the same sources. In more advanced stages the basement membrane is broken and dislocated, the retiform tissue more closely infiltrated with leucocytes, and the internal surface ragged and uneven. A still more advanced degree shows the mucous membrane completely infiltrated with cells. The most important fact as to catarrhal appendicitis is, that all three

stages offer favorable foci for the attack of pathogenic bacteria, and the starting-point of an infectious peritonitis. On the other hand, by the union of the opposing surfaces, obliteration of the lumen of the tube may take place, by which it is rendered immune against further attacks. A natural cure has, in a word, been effected. The obliteration may be partial, producing stricture, beyond which a cystic distention of the tube, generally near the cæcal end, is not infrequent.

II. *Ulcerative Appendicitis.*—In this form the mucous membrane and submucous tissue are destroyed to various depths, while the peritoneum may even be perforated. It is often asssociated with a concretion or a foreign body. The latter is now acknowledged as much more rare than was formally supposed. The error was a natural one, owing to the close resemblance of fæcal concretions to seeds, grains of wheat, cherry stones, and even date stones, as the result of a gradual moulding of shape and loss of water. The concretions are sometimes also the seat of deposit of lime salts. They may be multiple. They may be in the appendix a long time without producing harmful effect, the patient dying of other causes. The same is true of foreign bodies. Fæcal concretions are found in from 35 to 50 per cent. of cases, foreign bodies to much less frequent, say in 7 to 12 per cent.

III. *Infectious Appendicitis.*—This term is applied to a form of appendicitis which is always associated with bacterial invasion of the appendicular wall, and is, in all probability, due to it. It may be engrafted upon either of the two varieties just described, or it may arise *de novo*. In the former event it starts in the abraded or ulcerated surface described, or in the interior of the appendix wall. It is commonly associated with necrosis or gangrene of the wall, but may prove fatal before the necrosis sets in. The appearances vary greatly. They may be limited to a mere point, scarcely visible, and between this and the sphacelation of the entire organ there is every intermediate degree. The gangrenous organ is, however, usually enlarged and distorted. The virulence of the appendicular peritonitis is, however, just as great where there is no necrosis. One-half of all cases are believed to be of the infective form. The peritonitis, which ensues on perforation of the appendix, is also infective, resulting from the invasion of the peritoneum by myriads of bacteria in the fæcal matter set free at the time of rupture of the bowel. The minute appearances are as varied as the macroscopic, but Hawkins's summary of three more dis-

tinctive forms or degrees of infective appendicitis may be accepted as nearly correct. The first is characterized by necrosis of the muscular coats, the second by suppuration in them, and third by their infiltration with leucocytes and inflammatory exudation. The first is by far the most common. In all three bacteria are found in the mucous and muscular coats and all three are alike followed by virulent peritonitis.

The appendix may also be the seat, indeed is not a very infrequent seat, of tubercular ulceration even followed by perforation. I have lately seen a remarkable specimen of this kind in which no symtoms were present before death. So, too, a typhoid ulcer may form in the appendix and perforate with the formation of a tumor mass in the right iliac region. Follicular abscess may exist and occasion the usual symptoms of appendicitis. Actinomycosis has also occurred in the appendix with the formation of retrocæcal abscess and metastatic abscess of the liver.

Superadded to these conditions is often a localized or general peritonitis, the development of which, in the majority of cases, constitutes the attack of appendicitis. The result of the former is the iliac phlegmon or tumor. This occupies the right iliac fossa, and is variously constituted. It may consist of serous and cellular exudation, which mat together coils of small intestine and the cæcum. Or there may be a massive accumulation of cells and liquid, constituting abscess. Even the latter, as well as the more solid exudate, may be absorbed. On the other hand, the appendicular or perityphlitic abscess may rupture into the peritoneum, not infrequently producing fatal termination. The amount of pus varies. There may be a drachm or two, or a pint or more. More commonly there are two to four ounces. The pus is usually thin and very fetid; at times, it is thick, yellow and odorless. It may be mixed with fæcal matter. The pus may have escaped into the bowel, bladder or vagina, or externally at some point in the abdominal wall, as the navel, or groin as in a case of my own, or through the obturator foramen into the hip or thigh. The iliac muscle may be destroyed and the ilium bared. The abscess, usually in the iliac region, may be in the lumbar region or perinephritic, in the true pelvis, or under the liver. These very diverse sites are commonly determined by erratic situations of the appendix. There may be secondary abscesses of the liver by pylephlebitis or portal embolism. These may have all the terminations possible to hepatic abscess.

If general peritonitis supervene, there are added the usual anatomical appearances incident to this condition—flakes of lymph scattered over the intestines, binding the latter together, with pus in the flanks in varying quantity.

Etiology.—Of the three varieties of appendicitis described, the mildest form—catarrhal inflammation—is found unassociated with any demonstrable cause.

The second variety—ulcerative appendicitis—a form which includes many cases which have come to examination at operation, as well as at autopsy, is commonly associated with fæcal concretions or foreign bodies.

The third form—infectious appendicitis—which includes many instances of the most virulent inflammation may or may not be associated with mechanical agencies of the kind described, but the tissues of the appendix abound in bacteria, which, there is every reason to believe, are the sole cause of the inflammation in these cases.

While it may be that the milder catarrhal form of appendicitis is due to simple mechanical causes, or other similar, though less tangible agents, it may be reasonably doubted whether these are sufficient to induce the ulcerative form. Are the concretions and foreign matters not contagium-bearers, or, rather, do they not produce conditions favorable to the operation of pathogenic organisms already present and previously inactive, or convert harmless non-pathogenic bacteria into pathogenic?

A word as to the nature of the organisms which are responsible for the virulent forms at least. The *bacillus coli communis*, is a bacterium whose natural habitat is the colon of healthy individuals, cultures of which prove absolutely harmless when injected. Yet cultures of this same bacillus taken from cases of virulent appendicitis produce also corresponding virulence. Whence it may be inferred that in some way virulence is engendered in an otherwise harmless bacillus. There is good reason to believe that such bacilli may pass from the intestines to the peritoneum through an intestinal wall which is simply damaged, as well as through a perforation. Thus, many cases of so-called idiopathic peritonitis, or peritonitis where macroscopic examination revealed no evident lesion, may still be due to the bacteria of appendicitis from the interior of the tube. This has been actually demonstrated in some cases, and it is not unlikely that it will be found true of all cases thoroughly studied.

While in most instances the bacillus coli communis has been found in pure cultures, pyogenic bacteria have been found associated with it. The most important of these is the *streptococcus pyogenes*, and after this the *staphylococcus*, so that the existence of more than one infecting species may be admitted.

Predisposing Causes.—The most important predisposing cause of appendicitis is the appendix itself. An organ without use, and therefore undeveloped and feebly nourished, is correspondingly feebly resisting to all disease. As predisposing causes, too, must be considered certain influences which would formerly have been regarded as exciting causes, such as overeating, especially of unwholesome and indigestible food. It cannot be said that the precise mode of operation of such cause is certainly known. It may be that a hyperæmia or deranged circulation thus induced produces a condition favorable to the action of incessantly present bacteria. Similar is the effect of fatigue, cold and traumatic causes, such as blows and contusions.

Appendicitis is a disease of children and young adults. From 50 to 55 per cent. of cases occur under the age of twenty, and 30 per cent. between twenty and thirty. What bearing the fact that the appendix is longer in children and young adults has upon this, can only be surmised. Nearly 80 per cent. of all cases occur in males. It has been suggested that this is because the lumen of the appendix is larger in males, and therefore more liable to receive fæcal or foreign matters. Attacks have occurred, however, in the first year of life and as late as seventy-six. More cases occur in summer than in winter. Occupation has no effect in exciting it, but once established, recurring attacks of appendicitis are more frequent in men who do heavy work, such as porters and carriers, or men who stand long each day on their feet.

Symptoms.—Simple catarrhal appendicitis is often unattended by any symptoms whatever. The same is true of many cases of ulcerative appendicitis before the peritoneum is reached in the invasion. Other symptoms are more or less vague, though doubtless are present but overlooked. The infecting variety, consisting, as it does, in a simultaneous involvement of all the tissues gives rise promptly to serious symptoms. In point of fact, we know appendicitis to be present chiefly by the symptoms of the resulting peritonitis, local or general. Though in a majority of instances a first attack is a mild one, yet no one knows

at the onset whether this is going to be the case or not. Furthermore, it is often impossible to say when suppuration has taken place. The supervention of general peritonitis is, however, usually attended by unmistakable symptoms.

The first symptom is invariably pain, *sudden pain*. Its location at first is not constant. It may be anywhere in the abdomen. Most frequently, perhaps, it is in the neighborhood of the umbilicus. At other times it is in the epigastrium; at others, diffuse. It is intermittent, or at least remittent. Usually, within the first twenty-four hours, it settles itself in the right iliac region, where it remains. It may then be mild or severe, more frequently it is moderately severe. Even at this stage its location is not always constant. It has even settled in the left iliac fossa, under the liver or beneath the spleen, anomalous situations for the appendix. This pain is increased by coughing or taking a long breath, or turning over on the side.

As constant as pain, is *tenderness* in the right iliac region, or if the appendix happens to be placed in one of the unusual situations named, it will be in that situation. Rather strong pressure may at times be necessary to elicit it, but usually moderate pressure suffices. Its extent varies. It may occupy the whole lower quadrant of the abdomen or may extend up to the costal margin and around into the flank, but the seat of maximum tenderness is oftenest a point known as McBurney's—a point which is at the intersection of a line drawn from the anterior superior spinous process of the ilium to the umbilicus and another along the right edge of the rectus muscle. It is one and one-half to two inches from the anterior superior spinous process of the ilium. The patient almost invariably assumes the dorsal decubitus, often with the right leg drawn up, because of the relief thus afforded.

The third cardinal symptom, if the patient come under notice sufficiently early, is *rigidity* of the right *rectus abdominis* muscle and other muscles overlying the focus of inflammation. This may be associated with a slight distention of the entire abdomen. This primary tenseness, after two or three days, may be substituted by a *tumor*. The latter varies in size and shape, but is more commonly oval and about as large as a hen's egg, with its longer axis parallel with the upper part of Poupart's ligament. It may be much larger, occupying also the whole lower left quadrant and extending upward and back-

ward into the flank, while its shape may be quadrilateral or triangular. It varies in consistence. Its composition has been described in considering the morbid anatomy of the disease.

There is usually *impairment of resonance* to percussion over such a tumor, though less than might be at first expected. This is because we are really percussing over hollow organs, though matted together by exudation. At times, however, there is a duller note, while at others it may even be natural. In the latter event the tumor is small. Indeed, tumor may be altogether absent, but this can never be said of tenderness.

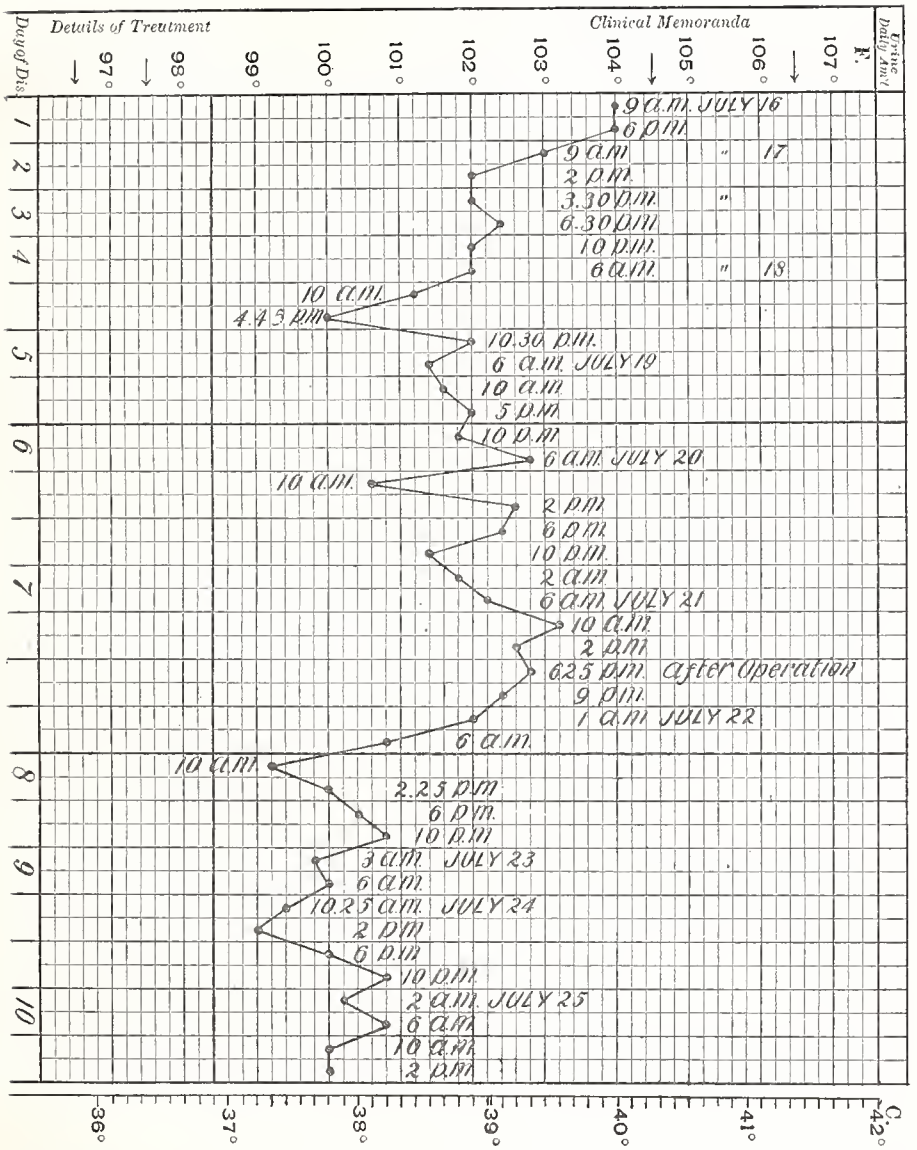
Vomiting is a symptom of more or less frequency. It is commonly regarded as reflex, and is variously severe. The matters vomited are first the gastric contents, with the evacuation of which it usually ceases, though it may recur with the event of perforation or rupture of abscess. If more prolonged, the vomited matter becomes greenish. Many so-called "bilious attacks" of past times have really been attacks of appendicitis.

Constipation is present in a decided majority of cases from the beginning of the attack. It is due to paralysis of the bowel, and may be so obstinate as to simulate obstruction of the bowel, being even attended at times with stercoraceous vomiting. Indeed, appendicitis has been often confounded with obstruction. On the other hand, there may be diarrhœa, recurring with each successive attack. There is loss of appetite; the tongue at first may be natural, but later becomes more or less coated, and in advanced stages dry.

There is always *fever* at the outset, the temperature 102°, 103° and even 104° F., rarely higher, after which it gradually falls, reaching the normal in five to seven days in favorable cases, which terminate in resolution. The pulse rate corresponds with the degree of fever, but its force and volume vary with the patient's strength. Should suppurations take place, the temperature continues with but slight fall, or may even rise further. (See Temperature Chart No. 1, appended.) *Suppuration may, however, be unattended with fever.*

Nor does a sudden fall of temperature always mean the establishment of convalescence. Not very rarely the event has a widely different meaning. It means that instead of convalescence perforation has taken place. It is extremely important that this fact should be realized. More than once have I known the physician to have

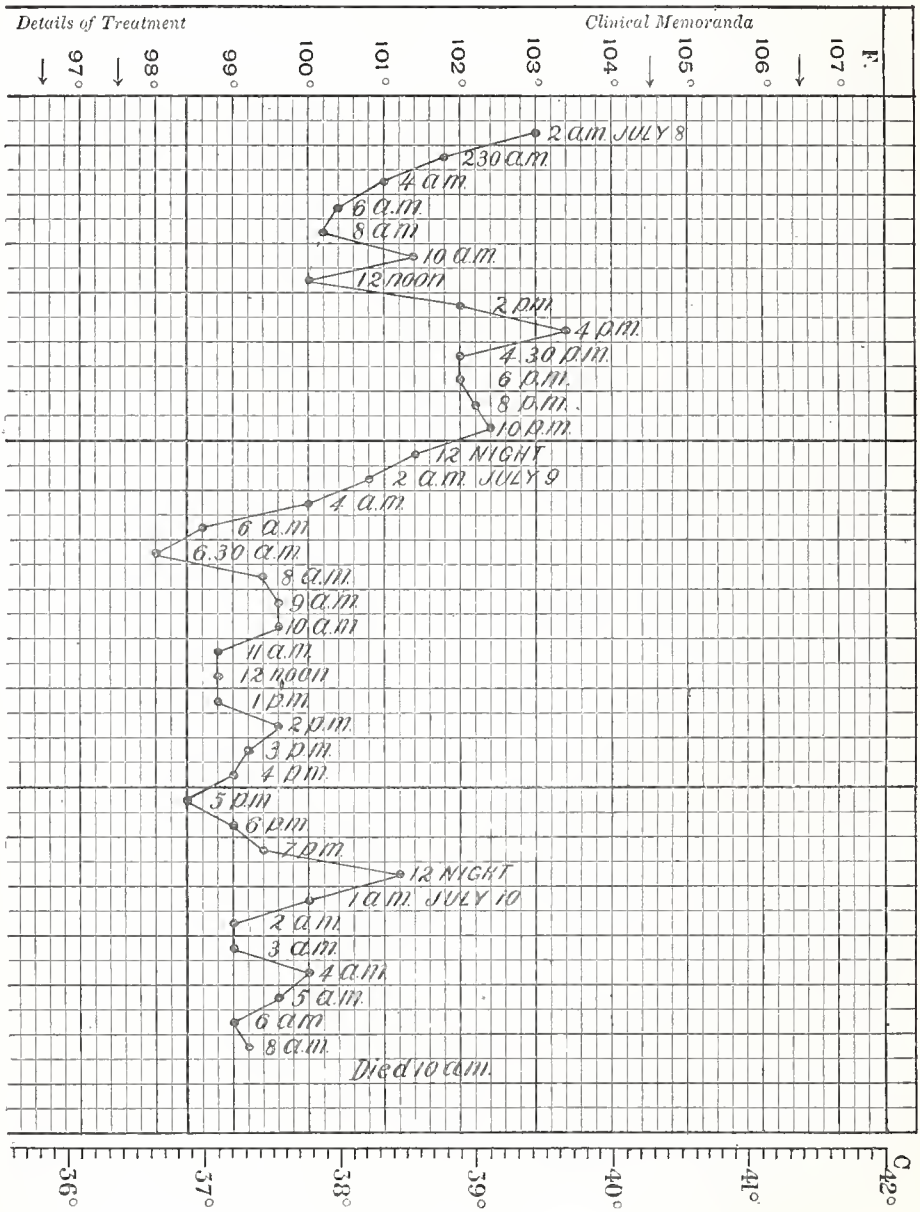
TEMPERATURE CHART NO. I.



The patient was first seen by Dr. C. F. M. Leidy at 9 A. M. on July 16, the first day of the disease, when the temperature was 104° F. (40° C.). It continued the same at 1 P. M. the same day. It then began to fall, and by 2 P. M. the next day reached 102° F. (38.9 C.). By 4.45 P. M. of the fourth day, it had fallen as low as 100° F. (37.7 C.), after which it rose and fluctuated about 102° F. (38.9 C.), again rose reaching 103.2° F. (39.5° C.), at 10 A. M. on the seventh day, when the patient was operated on by Prof. John Ashhurst, M.D., and an abscess evacuated, after which the case proceeded to convalescence.

been misled by it. The following temperature chart illustrates such a case. Another even more unusual explanation of sudden fall of temperature is the rupture of a small abscess into the bowel.

TEMPERATURE CHART NO. 2.



Finally, too much stress cannot be laid upon the fact that there may be gangrenous appendicitis in the presence of normal temperature.

The urine is scanty, as is usual in fever, and quite frequently contains

an abnormal quantity of indican. It is rarely albuminous, unless there be high fever, when there may be the small albuminuria characteristic of fever. There it often irritable bladder and frequent micturition.

The expression of the patient varies with the severity of the symptoms, but seldom exhibits the anxiousness characteristic of peritonitis, unless the latter actually is present in consequence of perforation or abscess rupture.

Is there any surer information of the event of *suppuration* than that furnished by the temperature as discussed? Fluctuation will, of course, be thought of, but this is rarely obtainable on account of the depth and mode of distribution of the pus. The pus may come to the surface and be thus recognized, but not often; and, furthermore, a case that has been allowed to proceed to this degree at the present day has not been properly handled. The rigor and sweat, such valuable evidence of the occurrence of suppuration under other circumstances, is, as a rule, wanting in appendicitis. Rapid growth of the tumor and the attainment of large size in a short time point to suppuration. But the most valuable sign is *the presence of exquisite tenderness over the focus of inflammation.* Continued high temperature is significant, though it may be wanting. Fully formed abscess has been found as early as the third day. More commonly six to eight days elapse before a diminished tenderness and slight decline of swelling point to this formation. Appendicitis allowed to go on to suppuration, *i. e.*, not relieved by operation, usually terminates by rupture into the peritoneum, followed by general peritonitis and death. The event is variously delayed by the extent and toughness of the protective adhesions which may have formed about the abscess. A few abscesses rupture into the bowel, thus saving the patient's life. Two or three cases in a hundred are thus saved. The fæcal fistula, incident in this termination, usually closes eventually, though not always. A smaller number of abscesses, especially if deeply situated in the pelvis, break into the bladder. The termination in these cases is less favorable, 50 per cent. being fatal. A few also break through the groin, followed by recovery. Lumbar abscess and perinephritic abscess must be mentioned as possible terminations, also infiltration of the abdominal walls and tissues of the thigh, pylophlebitis and hepatic abscess.

General peritonitis also ensues on perforation of the appendix, and

as well on the infectious invasion of the peritoneum already referred to as frequently incident to simple catarrhal and ulcerative appendicitis. The symptoms of the resulting general peritonitis are those peculiar to this disease, when suddenly induced by other causes. (1.) Diffuse pain as contrasted with pain localized in the right iliac region ; pain of extreme severity. (2.) Generally distended and tender abdomen. (3.) Moderate fever, which, succeeded by normal or subnormal temperature, already alluded to as often misleading the physician. (5.) Rapid and feeble pulse. (6.) Dry and coated tongue. (7.) The phenomena of collapse, *i. e.*, cold, clammy skin, feeble pulse, anxious expression, death.

RECURRING AND RELAPSING APPENDICITIS.

These terms are applied to cases of appendicitis which repeat themselves after a first attack. They are sometimes interchangeably used. The term chronic is also apt. Cases are called *recurring* which recur at considerable intervals, as some months, or a year or more ; *relapsing*, when the attacks are very close—at intervals, say, of one or two weeks, so as to make the attacks almost continuous. In the former, to which attention was first called by William Pepper in 1883, it is reasonable to believe that the patient has recovered in the interval of the periappendicular peritonitis, while the appendicitis, catarrhal or ulcerative, has continued, or there exists a cystic appendix as an exciting cause. In the relapsing form it seems likely that there has not been complete recovery in the interval. Certain it is that one attack predisposes to another, so that, in at least 23 per cent. of cases observed, according to Hawkins, and 44 per cent., according to Fitz, it is found that there have been previous attacks. The symptoms of a recurrent attack are the same as those of a primary one. In many cases the interval between the attacks is passed in comparative comfort ; in others, there is no small amount of suffering in the situation of the appendix.

Complications and Sequlæ.—The most important complication is obstruction of the bowels, by which is not meant the obstinate constipation so often met as an early symptom of appendicitis, but a true obstruction, the direct consequence of constriction by adhesions developed in the course of the peritonitis. Many striking cases confirmed by autopsy are related, and it is an important cause of death

in fatal cases, while operation frequently discloses conditions which could easily have produced obstruction.

Other complications are hepatic abscess from pylophlebitis due to thrombosis and even embolism of branches of the portal vein; also phlebitis of the right iliac vein. In abscess of the liver the diaphragm has been perforated, producing empyema and pyopericardium. Pyæmic abscesses elsewhere in the system, including the brain and lungs, have also been found in rare instances. Fæcal, bladder and umbilical fistulæ have been referred to. Fatal hemorrhage has also resulted from necrosis of the walls of the iliac vessels. Appendicitis may occur in a hernial sac.

Diagnosis.—The diagnosis of many cases of appendicitis is easy, and becomes more so as experience increases. A certain number of cases have to be carefully weighed, and in a few diagnosis is extremely difficult. Sudden pain becoming localized, tenderness and rigidity in the right iliac region are three symptoms which, if present, point almost unmistakably to appendicitis. The cases of difficult diagnosis are those in which these symptoms are wanting or are in unusual situations. But, in truth, these symptoms are less frequently absent than supposed. More frequently they are not looked for because there is very little to draw attention to them. A rule should, therefore, be made to examine carefully for them in any person subject to gastro-intestinal attacks, however induced and however manifested. It is certain that some cases of so-called catarrhal enteritis are really cases of appendicitis.

Differential Diagnosis.—Intestinal obstruction has been mentioned as a condition with which appendicitis has been sometimes confounded. The special symptoms of the various causes of obstruction, whether those of strangulation by bands or twists, by intussusception or by tumor or foreign body, should be recalled. Especially charasteristic of obstruction is the absence of fever, unless the patient lives long enough to permit peritonitis to be set up. The pain in obstruction is more intermittent at first, and though, like that of appendicitis, it may be anywhere in the abdomen, it is not likely to localize itself in the right iliac region. The constipation is more complete in obstruction, and even the passage of flatus is usually absent. The vomiting, also, is more severe and stubborn, and is more likely to be stercoraceous. There is more general distention of the abdomen, and limited tenderness is less easily differentiated. Intussusception occurs more

frequently in children younger than those subject to appendicitis, and is often attended with bloody discharges which never occur in appendicitis, while a tumor may often be felt on examination *per rectum.* Strangulation by bands or twists is more common in adults. Malignant growths, causing obstruction, are usually in the left iliac region, although cancer of the cæcum is to be remembered as a disease of the right. Its slower development distinguishes it from appendicitis.

Typhoid fever may be confounded with appendicitis, and I have more than once been startled in the course of a typhoid fever, by the thought that I might be dealing with an appendicitis, especially where there has been tympany and prolonged tenderness in the right iliac region; but one has, as a rule, only to recall the mode of beginning of the attack, the gradual development of the fever, its greater intensity and peculiar diurnal variation, the spots at the eighth day, to be reassured in the majority of instances. I recall one case of typhoid fever terminating in perforation, in which was simulated very closely even the iliac tumor of appendicitis. I may add that I do not think the typical spots in typhoid fever are ever closely approached by anything similar in appendicitis, though the event of suppuration is said to be sometimes indicated by an eruption. On the other hand, there is nothing to prevent typhoid fever and appendicitis from accidentally coinciding.

A question which one would naturally expect to give rise to difficulty is that, between appendicitis and the pelvic affections of women, such as an abscess around a fallopian tube or a pyosalpinx itself, yet mistakes from this cause seem to have been rare; and when we remember the deeper situation of the latter, and their usually easy recognition *per vaginam*, it is not so surprising.

Many cases of acute appendicitis were formerly mistaken for bilious colic and acute indigestion, but the latter are unaccompanied by tumor or tenderness, while the vomiting is more stubborn and the vomited matters differ. Entero-colitis occasions colicky pains, but there is no localization, while there is diarrhœa with mucous stools. It will be remembered, however, that these symptoms sometimes attend appendicitis.

In hepatic colic the pain is higher up in the region of the gall-bladder, while jaundice is almost invariably present, and sometimes pain under the left shoulder. In nephritic colic the pain extends

from the lumbar region into the groin and testicle. A floating kidney with twisted ureter is movable as contrasted with the iliac tumor of appendicitis; there is flattening of the corresponding lumbar region, while sudden relief to symptoms which characterize the untwist is altogether peculiar. The presence of blood in the urine is conclusive of the renal origin. In pyonephrosis there is tenderness in the region of the kidney, as well as pus in the urine. Perinephritic abscess occasions tenderness in the lumbar region, while the pain radiates into the groin as in nephritic colic. It is to be remembered that perinephritic abscess may be occasioned by suppurating perityphlitis where the position of the appendix is posterior to the cæcum.

Appendicular colic or neuralgia of the right iliac fossa is a vague condition of pain in this region, which has been ascribed to peristaltic contraction of the appendix, constituting an effort to expel fæcal pellets, but of which no proof is afforded, operation in several cases failing to discover anything abnormal.

Prognosis.—It is a difficult matter to consider fairly the prognosis of appendicitis, or rather of the perityphlitis growing out of disease of the appendix. For, if we separate the cases which do not go on to suppuration, recovery is apparently the rule. Thus, out of one hundred and ninety cases collected by Hawkins, none died. Again, of cases treated by section and drainage after suppuration has set in, fully 25 per cent. die; while if general peritonitis supervene, 75 per cent. die.

On the other hand, it is impossible to say of any case, however mild, that if left alone it will not terminate in suppuration, while a large number of cases still perish, because of imperfect diagnosis and delayed operation.

Treatment.—As soon as the diagnosis of appendicitis is established, indeed pending its settlement, a competent surgeon should be associated with the physician, for the reason that in the vast majority of cases operative treatment is sooner or later demanded, while the hour for such treatment is best settled by daily conference. The course of cases of appendicitis is often very delusive, and the surgeon who operates frequently is likely to have seen more cases than the physician. The diagnosis being established, operative treatment should be recommended, except in those cases where the disease is so far advanced as to make it unlikely that the patient will be saved by operation. My reason for this belief is, that while a majority of cases of

simple appendicitis may subside with rest, in a very large number, at least 25 per cent., the primary attack leaves the patient predisposed to another, at once more severe and dangerous than the first, while we have no guarantee that any attack will subside without suppuration, or, what is worse, without leaving the condition referred to, in which malignant inflammation or perforation may set in at any moment without warning.

It must be admitted that it is not always easy to lay down a rule by which operation shall be determined, for it is not only that we must know when to operate to save life, but also that we must know when not to operate in cases so severe that operation will be futile, and it is due the operation that it should be saved the approbrium of such futility. Certain it is, too, that in cases where operation is of no avail death will be hastened by it, the depressing effect of etherization co-operating to hasten the fatal end. Much difficulty is, however, removed when we decide to operate *without undue haste* in all cases so soon as the diagonsis is established, except where operation will evidently be futile. I say without undue haste. For, in many cases, it is plain that a few days' delay, if the patient is kept at rest, will make no difference in the result, while, if the inflammation is subsiding, a stage is being reached in which the operation is even less dangerous, since the united experience of surgeons goes to show that the mortality of operations between attacks is practically nil, while that immediately succeeding diagnosis in ordinary cases is nearly so. There can be no doubt, moreover, that excision of the appendix *after* a first attack is a safer procedure than *during* a first or any attack. Even where suppuration has set in it may be safe to delay operation for a day or two while the patient is held quiescent.

When, on the other hand, shall operation be omitted because it must be followed by a result inevitably fatal? In all cases where there is diffuse septic peritonitis, rapid pulse and leaky skin, constant vomiting and constipation, operation is generally futile. In such cases saline purgatives, stimulants diffusible and cardiac are indicated, and rarely, though rarely indeed, recoveries have taken place.

Medical Treatment.—Cases must occur, however, in which from various causes medical treatment is necessary. Operation may be declined even if urgently advised, while rarely a preparative medical treatment may be necessary previous to operation.

First of all, absolute rest in bed must be insisted upon as the first essential condition of abatement of the inflammation. Many a fatal case would have been saved had this injunction been carried out.

Next, relief of pain is demanded. For this purpose opium should be avoided except in extremest cases. Only when relief cannot be secured by the ice-bag, by poultices, by hot fomentations with mild counter-irritant, by mustard or turpentine or by leeching, may a minimum dose of morphia, one-twelfth or one-eighth grain, be given hypodermically. The objection to opium is well founded on the ground that it masks the presence of important symptoms which should be open to observation. In cases where operation is from any cause out of the question, counter-irritation by repeated blistering may be practiced, and excellent results were reported under the older treatment before operation became common.

The question of the propriety of giving an aperient is a delicate one, and must be left, for the most part, to the circumstances and the good judgment of those in attendance. The result may be very happy or mischievous. Cases may be so advanced or severe that the effect of a purgative may be to cause perforation or the rupture of an abscess, but in ordinary cases or cases of moderate severity an aperient may be useful to clear up a diagnosis, while it relieves pressure, depletes the bloodvessels and diminishes the danger of peritonitis. On the other hand, purgatives should not be aggressive and drastics should not be used. Perhaps a safe rule would be, "if there is doubt, do not purge." The best aperient is castor oil, followed, if necessary, by salines, and of these Rochelle salts or the solution of citrate of magnesium. If the stomach is sensitive, calomel in divided doses triturated with sugar of milk, is the best drug. Where there is reason to believe that suppuration has set in no purgative should be given, and, as a rule, operation should be prompt towards evacuating the pus and removing the appendix at the same time. In severe cases even enemata should be avoided as tending to favor perforation and rupture.

Nourishment should be purely liquid, and of liquid, milk is the best, though animal broths are not contra-indicated. It should not be an object to force food; indeed, only the minimum sufficient should be permitted.

Whatever preparation is deemed necessary for operation when decided on must be laid down by the surgeon.

RECURRENT HEPATIC FEVER.

Clinical Lecture Delivered at the Philadelphia Hospital.

By ROLAND G. CURTIN, M.D.

Reported by Dr. H. BALDWIN, Resident Physician.

The specimen which I bring before you this morning, is the liver of a patient who died in my wards from exhaustion, the result of advanced age and the effect of recurrent hepatic fever. This disease is also known as Charcot's fever, or French fever. It is characterized by recurrent attacks, simulating intermittent malarial fever. In the typical form there is a sudden onset, with chill and sudden rise of temperature, followed by sweating. There is pain over the liver, and there may be vomiting. Usually there is a gradually increasing jaundice. The temperature assumes an intermittent type, which continues for several days or weeks, when the symptoms disappear and the temperature becomes normal.

The disease is caused by irritation of the ductus communis choledochus, by inflammatory conditions, or by temporary occlusion on account of a biliary calculus or an inflammation of the gall-bladder.

The notes of this case are as follows :

J. L., aged seventy-seven years; female; white; single; born on the Isle of Man; occupation, dressmaker.

Family History.—Father died of paralysis; mother, from old age. One sister died from some hepatic disease.

Previous History.—Milder diseases of childhood. No other sickness.

Present Illness.—For the past twenty years the patient has been subject to attacks of chills, followed by fever, and often accompanied by jaundice. She sometimes has slight pain over the liver, but this is never severe. With these attacks she has had general malaise and occasionally nausea and vomiting. A year ago she was treated in this hospital for one of these attacks, in which she became deeply jaundiced. Since then she has had milder attacks, only one of which has required hospital care.

Present Condition.—On admission, September 23, 1895, the patient was emaciated. The skin and conjunctivæ were slightly tinged with yellow. The heart's

action was weak; no murmurs; the arterial walls were sclerotic. The lungs were apparently normal. The liver dulness extended from the upper border of the sixth rib to the costal margin in the mammary line. There was slight tenderness over the hepatic region. Examination of the urine showed no abnormality. The tongue was heavily coated, appetite poor, the bowels constipated. The patient was admitted just after a chill and she was much exhausted. Temperature, 102.2° F.; pulse, 112; respiration, 42.

She improved under treatment and the jaundice disappeared, but she continued to have an evening rise of temperature of from 100° to 102°. The temperature was for the first week intermittent, falling below normal each morning. The second week it became remittent in type. The pulse-rate was slow, being only about 67 per minute, while the temperature varied from 101° to 102° F.

On October 10th the patient had several slight chills. On the 11th she had severe chills with profuse sweating. There was no rise of temperature with the chills, the evening temperature being 98.8°. There was no jaundice and no pain over the liver. She failed rapidly and died October 12th.

The liver, as you see it now, is contracted and discolored by the hardening reagent in which it has been placed. The gall-bladder protrudes now not more than one-half inch beyond the liver margin. Before removal from the body it was entirely concealed behind the liver. The liver and gall-bladder were both adherent to the intestines. The gall-bladder is here shown filled with concretions. At this point, a small calculus is seen protruding from the opening of the common duct into the duodenum. This was doubtless the cause of the attack in which she died. The liver on section appears spongy and marked by small holes, which are the dilated bile vessels. These openings give the cut section of the organ an appearance like fermenting dough. There are biliary concretions found in the liver. The weight of the organ is 1430 grammes.

The pathological changes found in the other organs at the autopsy were, in the main, those of senile degeneration. The ventricles of the heart were dilated and the heart muscle soft and friable. The valves were competent, the aortic being marked by atheromatous deposits.

The lungs showed the lesions of chronic bronchitis with marked œdema.

The spleen was much enlarged; its weight, 510 grammes. Its capsule was thickened, very rough and marked by old, small granulations.

The kidneys showed some interstitial degeneration, with the formation of small cysts.

The walls of the arteries were sclerotic.

In connection with this case it is interesting to consider that of another patient whom I have recently seen. During an operation for floating kidney in the latter case, it was discovered that the gall-bladder was filled with calculi. A Murphy's button was introduced, connecting the gall-bladder with the intestine. After this operation over five hundred gall-stones were passed from the bowel. The patient has since become pregnant and has been delivered, with no complication arising from the previous operations.

The diseases for which hepatic fever is most often mistaken are malarial fever and fever of septic origin. The diagnosis from malaria is readily made by examining the blood for the plasmodium malariæ. The making of the diagnosis is also aided by the administration of quinine, which will check the disease if it is malarial. The diagnosis is further rendered clearer by the discovery of the gall-bladder protruding below the liver margin, but, as you see in the present case, it may be drawn up behind the liver so that it cannot be examined. The history of a previous attack of biliary colic helps to determine the nature of the disease.

The diagnosis is important, because if the presence of the calculi in the gall-bladder is recognized, the condition can be relieved by operation.

REPORT OF A CASE OF MALARIAL FEVER, WITH A STUDY OF THE BLOOD.

William Humphreys, forty-eight years; white; born in Scotland; painter; single, was admitted to the hospital October 8, 1895.

Fourteen years ago, while living in Wilmington, Del., the patient had an attack of malarial fever. This was at first tertian in type, later becoming quotidian. For three years after this he was subject to attacks of chills or "dumb ague."

Since then he has had no pronounced malarial symptoms, but he has never been well. He has been subject to dizziness, backache, pain in the legs and pain in the side, which he refers to the region of the spleen.

Two weeks before his admission to the hospital he spent a night at Coatesville, Pa. While there he had a severe chill. Since then he has had no chill, but thinks he has had continuous fever, and he has suffered from great prostration.

On admission his temperature was 104°, soon rising to 105° F. The examination of his blood at that time showed a great number of extra corpuscular hyaline bodies—freely motile. No other forms of malarial organisms were discovered.

On the day of his admission he was given quinine sulphate, gr. xxx, with repeated doses of magnesium sulphate. On the next day fifteen grains of quinine were administered; on the third, thirty grains, and then fifteen grains each succeeding day to the sixth.

After the third day his temperature remained normal and his symptoms entirely disappeared.

On the fifth day after admission a few crescentic forms of the plasmodium malariæ were found in the blood. At the end of the first week he was placed on a pill containing:

R.—Quinin. sulph. . . . . gr. ij.
Ferri pyrophosphat. . . . . gr. ij.
Ac. arsenios. . . . . gr. $\frac{1}{30}$.—M.

This pill was given three times a day. At the end of the second week after admission the patient's general condition was much improved. He still had no malarial symptoms, but, on examination of the blood, crescentic forms were found in quite large numbers.

The same treatment was continued during the third week, and at the end of that time the crescentic forms were still quite numerous in the blood.

The dose of quinine was then increased, so that he received three times daily:

R.—Quinin. sulph. . . . . gr. v.
Ferri pyrophosphat. . . . . gr. ij.
Ac. arsenios. . . . . gr. $\frac{1}{30}$.—M.

After three days he began to have symptoms from the quinine—a feeling of fullness in the head with headache and ringing in the ears. A careful examination of the blood was made, but *no malarial organisms were found*, and after that day no more could be discovered.

APPENDICITIS—REPORT OF A CASE.

By SAMUEL WOLFE, A.M., M.D.

For the notes of this case I am indebted to Dr. John C. Welch, at the time my resident physician.

A young English laborer, aged seventeen, with a good family history, except that a sister had died of cancer, and who had not had any previous illness, was admitted to the Philadelphia Hospital, into my ward, September 17, 1894.

He had some headache, and great pain and tenderness in the right iliac region, with a well-defined sausage-shaped tumor. There was no abnormal temperature, a somewhat coated tongue, and a fairly well-marked systolic murmur. He gave a history of having been attacked three weeks previously with severe abdominal pain, which localized itself in the right side, and having suffered about one day with severe headache and general indisposition. The pain, or at least tenderness, had continued with varying severity up to the time of his admission, but during a part of the time he had still worked.

For the first ten days he had frequent bowel-movements, but passed little at stool, and subsequently he was absolutely constipated.

During his first day in the hospital, Dr. Welch asked several members of the staff to see him, and they considered the case one of appendicitis. I saw him late on the second day of admission, and found such extreme tenderness in the right iliac region that he could hardly bear the lightest touch.

I ordered four leeches to be applied at once, to be followed by cold lead poultices, with a liberal quantity of laudanum added. An enema containing tr. assafœt, f℥ij; ol. terebenth., f℥ij; ol. ricini, f℥ij, in one pint of warm starch-water was ordered at once, to be followed by copious enemas of warm, soapy water; calomel, gr. ss, was ordered every two hours, and next morning magnes. sulphat., ℥j, in hot water. Instructions were left with Dr. Welch to turn the patient over to the surgeon in the morning for operation, if resolution had not taken place. The patient, however, was relieved of the tumor, and the tenderness was greatly allayed by next morning, and in two days he was entirely well. He was detained in the hospital as a precautionary measure for a few days longer, and then discharged.

A SMALL SERIES OF CASES OF LEAD POISONING, WITH SOME REMARKS UPON SATURNINE GOUT.

By FREDERICK A. PACKARD, M.D.

The short series of cases herein reported show some points of interest that separate them from the usual run of instances of saturnism as seen in hospital practice, and the features presented by them induce the writer to publish what seems like a group of cases too small to be of much value. Some of the cases are reported because of the presence of distinct gout—a symptom of lead poisoning that is certainly far less commonly met with in America than in England, and must be counted among the unusual manifestations of saturnism as it is seen in Philadelphia. Other cases are reported on account of the mental symptoms present, and although they are few in number they may, it is hoped, be of a little value to some investigator collecting data in regard to this interesting manifestation of poisoning by lead. The other cases are briefly reported because of the presence of the lead discoloration, not only on the gum, but on the lip. These also are, unfortunately, few in number, but so frequently in text-books is the existence of such a distribution entirely unnoted, and so unusual is its occurrence, that the writer only learned of its recognized existence when looking up the literature of the subject upon observing the first case seen by him. As it is not more frequently present in cases applying for treatment, and as it is so little dwelt upon in any text-books or monographs save for the monumental work of Tanquerel, the writer has hoped to draw to this sign the attention of some who, like himself, had never appreciated the possibility of its existence.

The following cases of gout, apparently due to lead impregnation, have been under the writer's care:

Case I.—T. L., white, aged forty years; born in England; a resident in the United States for twenty years; admitted to Ward VI. of the Philadelphia Hospital on September 22, 1894; history dated October 22. His mother died of asthma; his father of old age. Six brothers and one sister are living and healthy. One sister died of gangrene of the foot, the nature of which cannot be ascertained.

He has been a glass-cutter and polisher during the whole of his adult life, working in an atmosphere filled with dust from the "lead dross" used as a polishing agent. The room in which he worked was small and poorly ventilated. His habits have always been temperate as regards alcohol and he denies venereal infection.

Ever since he began his present work he has been subject to frequent attaeks of vomiting and constipation, with the presence of a sweet taste in the mouth. After drinking freely of milk these attacks would gradually pass off and be succeeded by a spell of diarrhœa. He has never had any sign of lead-palsy. In 1890 he had a severe attack of pain, accompanied by redness and swelling in the metatarso-phalangeal joints of the great toes, extending upward over the insteps. The proximal phalangeal joint of the middle finger of the right hand was likewise the seat of boring pain, swelling and redness. After the acute symptoms subsided, desquamation occurred over the affected joints. For three months he was unable to work, owing to the pain and stiffness resulting from this attack. Since that date he has had a return of the pain, swelling and redness in the same joints upon about six occasions. Two years ago he had an attack of severe colic, with marked constipation and intense pain in the head, back and legs. In September, 1893, he began to be troubled with double vision, and soon after the onset of that symptom became suddenly blind in the course of an afternoon. For two weeks he could see nothing; but at the end of that time improvement began and continued until September, 1894.

He was in the Philadelphia Hospital in December, 1893, on account of an attack of colic, with pains generally distributed throughout the body. In June, 1894, he was discharged from the hospital and returned to work, but could do only a little labor and was soon compelled to desist from the attempt, owing to headache, generalized aching pain and weakness.

He was re-admitted to hospital on September 22, 1894, on account of increasing weakness, headache and progressing loss of vision.

Upon his last admission his condition may be summarized as follows: He was quite weak, pallid, and rather poorly nourished. The pupils were normal and responded well to both light and accommodation. The tongue was clean, teeth in fairly good condition, marked narrow blue line on margin of both gums. Examination of the lungs was negative. The heart showed no abnormal signs, save for accentuation of the second sound at the aortic area. Abdominal examination negative. The gait was normal; there was no inco-ordination, no ankle-clonus; knee-jerks exaggerated on both sides equally. No evidence of loss of power in arms or hands was noted. The urine was scanty (eight to twelve ounces in the day), was pale in eolor, acid, specific gravity 1010, contained a marked amount of albumin ("one-eigth by bulk"), hyaline and pale granular casts.

He stated that his mind was less active than before his illness began. After admission he had marked looseness of the bowels, upon subsidence of which he made no complaint, save of diminution of vision and of weakness, and presented no abnormal signs, save for the blue line upon the gums and the evidences of renal disease. The latter became rapidly less marked, as is shown by the note made on October 15th, that the urine had increased to a daily average varying between forty and seventy ounces; that on the above date it had a specifie gravity of 1015, contained a mere trace of albumin, no casts, but a deposit composed of mixed urates and uric acid crystals.

Upon October 15th, Dr. Gould kindly examined the eyes for me and reported as

follows: There is no change in the fields of vision, the ocular movements and pupillary action are normal. There is a chronic central choroiditis, but no evidence of albuminuric retinitis.

Upon the same day the blood was examined and found to contain 2,100,000 red-blood cells to the cubic millimeter, the hæmoglobin contents being 48 per cent. During the latter part of October he had constantly recurring attacks of cephalalgia, usually frontal, and of pain vaguely located in parts below the knees. There was much depression of both mind and body.

On November 6th he had severe pain in the proximal joint of the right middle finger, which became swollen, reddened and exquisitely tender. Two days later a similar condition was present in the metatarso-phalangeal joint of the left great toe. Upon the 10th of November the whole left foot was involved, the pain, swelling and redness extending upwards to the malleoli. Upon the 12th the pains were less acute, and an examination of the urine gave the following result: Acid, specific gravity 1020, albumin (about one-fourth by bulk on boiling), many granular casts and uric acid crystals.

The note of November 28th states that the patient had recovered entirely from his attack of gout, but that there was still slight desquamation on the surface of the parts affected.

The cephalalgia continued, and was somewhat relieved by the application of a cantharidal blister to the nuchal region. (Serum from the blister failed to deposit crystals of uric acid upon a thread after the addition of acetic acid to the fluid; but for unavoidable reasons the test was not accurately performed.) The most permanent relief of headache was afforded by the occasional use of the hot-air bath.

The patient has now (January, 1895) been in about the same condition for the past two months. He is pallid, languid, much depressed in spirits, suffers much from frontal headache, and has occasional twinges of pain in the small joints of both hands and feet. There is a persistent faint trace of albumin in the urine, but no casts can be found on careful search.

A recent examination of the eyes, kindly made by Dr. G. E. de Schweinitz in the latter part of January, 1895, furnished the following results: Pupils normal in reaction. *O. D.* Partial consecutive atrophy of disc with some haziness of margins and much white tissue along the vessels (perivasculitis). Fine granular changes in choroid at macula. *O. S.* Similar to right. Vessels about normal in size. Vision, $\frac{1}{5}$ in each eye.

Diagnosis.—Retrogressive neuritis with perivasculitis.[1]

Case II occurred during the writer's service at the Episcopal Hospital during the summer of 1892:

Case II.—Richard B., white, aged thirty-eight; hospital number, 1429; born in the United States; admitted to hospital on September 8, 1892; discharged September 26, 1892

His family history was absolutely negative as regards any predisposing factor.

[1] The patient left the hospital in the latter part of September, 1895, and again went to work at his former occupation. At the end of two weeks he had to abandon it owing to the occurrence of general pains, especially marked in fingers and toes. He is now (December, 1895) convalescing from a recent attack of gout of the great toe-joints, which manifested itself a few days after his readmission to the hospital in the latter part of October.

He is married, but has had no children. He had the ordinary diseases of childhood, but recovered perfectly from them and had been usually healthy. He had gonorrhœa when a young man, but denied other venereal disease. He stated that he had never been a hard or steady drinker of alcohol.

He had been a painter since the age of twenty-two. He had never had any manifestation of plumbism, save for chronic obstinate constipation and an attack of colic in 1883, until eighteen months before his admission to the Episcopal Hospital. At the time mentioned he had an attack of colic for which he was treated in a hospital for six weeks, had a blue line in his gums, but had no "wrist drop." He had for some years had occasional attacks of "aching pains" in the extremities, but had had no evidence of organic change in any of the articulations. On close questioning it was found that upon his discharge from the hospital after his attack of colic he had some thickening of the distal inter-phalangeal joint of the left index finger and the second joint of the middle finger of the right hand, and also that for many years he has occasionally lost control of his brush, owing to flexor spasm in the right hand. On September 1, 1892, seven days before admission to the Episcopal Hospital, he was suddenly attacked by pain in the metacarpo-phalangeal joint of the little finger of the right hand. Two days thereafter the entire finger became much swollen and intensely painful. In four more days the right elbow and shoulder became extremely painful, the elbow swelling and becoming of a bright red color.

Upon admission to the hospital the pain in the right upper extremity was so intense that the clothing could not be removed until his suffering had been lessened by the hypodermic injection of morphine.

Examination showed that he was a fairly well-nourished man of medium height. The face expressed severe pain with frequent sharp exacerbations. The tongue was pale and lightly coated with white fur. On the upper and lower gums was a distinct continuous narrow blue line, most marked in the projections between the teeth. Physical examination of chest and abdomen revealed no morbid change. Examination of the urine negative; specific gravity, 1020. Temperature upon admission, 99⅗° F.; pulse, 72; respirations, 20.

On helix of left ear were two small shot-like tophi of yellowish white color, apparently on the point of bursting through the epidermis. On the right upper eyelid was a similar yellowish-white tophus. The whole right hand is reddened, puffy and extremely tender to the touch, the skin being apparently tightly stretched and shiny over the dorsum. The part most swollen, reddened and tender was over the metacarpo-phalangeal joint of the right thumb. Another area of more intense trouble is around the first inter-phalangeal joint of the middle finger. The right elbow is swollen and reddened, and over the olecranon the skin is quite shiny. The shoulder shows no abnormality, save for extreme sensitiveness to touch or motion.

The patient was at first put upon salicylate of sodium, with some relief from pain, but upon the next day, owing to headache and ringing in the ears, this was substituted by the wine of colchicum and the whole arm was enveloped in cotton-wool. Upon the day after admission the pain was much less, but the temperature reached 101° F. Upon the 12th of September all pain had disappeared and the diffuse redness and tension of the skin gave place to punctate redness and slight desquamation over the two most intense points of trouble. Potassium iodide was then given in place of the colchicum.

Improvement continued until September 23d, when the right metatarso-phalan-

geal joint, the left knee and both shoulders were attacked by boring pain. This subsided under the continuous use of potassium iodide and the application of cotton-wool to the parts, and no further symptoms occurred prior to his discharge upon September 26, 1892.

In the next year the patient was again admitted to the Episcopal Hospital during the service of my colleague, Dr. D. J. M. Miller, who kindly notified me of his return to the ward and asked me to see him with him upon one occasion. He had been suddenly attacked with loss of power in the arms and legs. For several hours before admission he had been wildly delirious. When I saw him there was almost complete palsy of the whole body with combined wasting and spasticity in all of the extremities. After a severe illness he recovered perfectly of his nervous symptoms under large doses of potassium iodide, and was discharged cured.

I wrote in the early part of February of this year, asking him to come to see me and report upon his condition since I had last seen him. He gave me the following brief account of himself: He seemed to recover perfectly from the illness for which he was last treated at the Episcopal Hospital, and had returned to work, although cautioned against continuing to work with lead. He has never felt perfectly well since his first attack of gout, his feet giving him much pain after he has been standing for any great length of time. On November 16, 1893, he entered and was treated at the Hahnemann Hospital for three months for gout.

He is now fairly well-nourished—lips of good color, skin not markedly pallid. The pupils are equal and natural, no arcus senilis. Tongue slightly coated. About the insertion of two of the teeth on the lower jaw are a few dots of doubtful bluish-black staining. On the helix of the left ear are two tophi resembling in appearance those remembered as present on his admission to the hospital two years and four months ago.

On the right hand the first inter-phalangeal joint of the middle finger is much enlarged, chiefly laterally and dorsally, and the finger is held partially flexed; extension and flexion, either active or passive, being impossible, owing to locking of the broadened and thickened articulating surfaces and to the periarticular thickening. On the ulnar side of the right wrist there is a distinct osteophyte. On the left hand the two inter-phalangeal joints of the index finger are enlarged and nodular, as is also the last joint of the little finger. There is no sign of active inflammation in any of the affected joints. Movement of elbow and shoulders unimpaired and contour well preserved.

Station is normal both with open and closed eyes. Gait slightly halting, but evidently from articular stiffness. There is no notable wasting, although the muscle tone throughout the body is below par. The knee-jerks are very active upon both sides, but are not exaggerated beyond the normal limits. Ankle-clonus could not be elicited. In the upper extremities the reflexes are normal. There is no alteration of sensibility to touch or pain. Temperature-sense and electrical condition of muscles could not be determined at the time of examination. The eye-ground presented no deviation from normal.

Case III., apparently to some extent due to plumbism, was seen twice by the writer in the out-patient department of the Pennsylvania Hospital, in 1893.

The patient was a white man, aged forty-eight, a native of America; hospital number, 1904. His family history was negative, save for the fact that his mother

suffered from "rheumatic gout." He has been a quite free drinker of spirits. He had been a printer for twenty-eight years. He denied venereal disease. Four or five years before he had had lead colic and wrist-drop. He had a sharp attack of pain at the base of the left great toe three years before coming under observation, and since that time had suffered from frequent twinges in the same foot. A second attack occurred in June, 1891, in the same toe. The attack for which he sought treatment had begun on the day before he presented himself, coming on suddenly during the afternoon, although he had for some time been apparently in his usual condition of health.

He was a moderately well-nourished man, but the skin and mucous membranes were quite pallid. No tophi were present, nor had he any skin lesions. No blue line was present upon the gums. The arterial tension was quite high, but there were no abnormal cardiac signs, the second aortic sound alone being accentuated. The left metatarso-phalangeal joint on the great toe was much swollen, reddened and tender. Pain was complained of up the leg as far as the knee, but there was no outward sign of trouble, save at the base of the great toe.

Rapid improvement followed the administration of a combination of lithium citrate and potassium carbonate.

The following cases of encephalopathy from lead intoxication have been observed:

CASE IV.—A. K., white, aged fifty; born in Germany; was admitted to the Philadelphia Hospital on October 8, 1894. He came to America in September, 1893.

His parents died of cholera in Germany. Five brothers and four sisters are dead, all of either typhus fever or measles.

He was a plasterer in Germany, but almost as soon as he landed in this country he went to work in a white-lead factory. His work kept him in the "packing-room," in the midst of a cloud of white-lead dust.

Except for an attack of small-pox, he was always a healthy man until the time of his undertaking his new occupation. He has used tobacco and alcohol to excess.

In June, 1894, after having been working in lead for eleven months, he had an attack of colic, accompanied by headache, tormina and constipation. There was no palsy of the extensors of the wrists. The attack ended with diarrhœa after an illness of five weeks. Recovery, however, seems to have been perfect. In September, 1894, he resumed work, but on the twenty-eighth of that month he came home from work owing to a return of the colic. It was at that time noticed by his wife that he "acted queer." On October 3d he was suddenly seized by a convulsion, in which he bit his tongue and lost consciousness. The convulsion was of short duration, but it was repeated three times in the succeeding forty-eight hours. He then became violently maniacal and was admitted to the "Detention Ward for the Insane" at the Philadelphia Hospital on October 8th. On admission he was in a state of violent maniacal excitement.

Physical examination on admission showed nothing abnormal, save for the presence of a very distinct blue line upon both upper and lower gums at their margins.

He was transferred to the general ward after a short stay in the detention ward. His physical condition remained unchanged. The urine was acid, of high color, 1028, showed no albumin or sugar, but under the microscope there were found urates and uric acid crystals. The blood examination showed: red blood-cells, 3,600,000; hæmoglobin, 60 per cent.

On free purgation with Epsom salts and a liberal use of the iodides the mental symptoms quite rapidly diminished, but he for some days remained rather talkative and excitable.

He was discharged, at the request of his wife, much improved as regards the mental symptoms, on the fourth day after his removal to the general medical ward. Just prior to his discharge the red blood-cells numbered 4,200,000 per c.mm.; hæmoglobin, 70 per cent.

CASE V.—John K., white, aged twenty-six years; was admitted to the Pennsylvania Hospital on October 27, 1894. He was born in Ireland, and came to America in April, 1893. His family history was negative. He was always a strong and healthy man, never having been confined to bed by illness. Drinks whiskey, but he says that he never gets drunk. Does not use tobacco. Denies venereal disease.

He began working in lead six weeks before admission to hospital, but two weeks later had to discontinue, owing to an attack of abdominal pain. His work consisted in shoveling lead into a "mill." The atmosphere of the place in which he worked was full of lead dust. He used no precautions against the ill effects of lead, such as were employed by some of his co-laborers.

As stated above, two weeks before his admission he was seized with a severe attack of abdominal pain of a colicky nature, accompanied by vomiting and obstinate constipation. These symptoms persisted until his admission to the hospital.

On admission his temperature was 98° F.; pulse, 116; respiration, 20. He was intensely pallid, the skin being of a dead-white color and the visible mucous membranes almost as pale as the skin. The tongue was pale, clean and dry. At the insertion of the teeth into the gum in both jaws there was a well-marked, sharply-defined blue line about a millimetre in depth. Physical examination of the thorax showed no abnormality. The abdomen was prominent rather than scaphoid. The urine showed no abnormal constituents; specific gravity, 1030.

He was tossing about the bed from the severity of his abdominal pain, but the mind was clear and there was no evidence of paresis of any muscles.

Under routine treatment by Epsom salts and morphine and atropine he seemed to be rapidly recovering, but upon the 4th of November, eight days after admission, he first began to have mild delirium, shown by his almost continuously talking to himself. It was at first thought that he was talking in his sleep, but upon approaching him it was found on several occasions that the patient's eyes were wide open and that he answered questions rationally after a quick start upon being first addressed. Careful examination revealed no sign of inflammatory trouble in the brain or its envelopes.

During the night of November 5th he became violently delirous, and had to be restrained by straps. He talked loudly and disconnectedly, and would occasionally cease talking and whistle vigorously for a time. He was put upon large doses of potassium iodide.

On the next day the delirium continued, but was somewhat less noisy. On November 7th he was much quieter and could be quite readily calmed. Improvement steadily continued as regards the mental condition; he became entirely rational by the 11th of November and was allowed to go home, against advice, upon the 15th of the month.

Four cases have been seen by the writer during the past winter where there was present a blue line on the mucous membrane of the

lip as well as upon the gum. In one the line was upon both lips, in two upon the lower, and in one upon the upper lip. In all the line about corresponded in position with the line upon the gums—that is to say, the two blue surfaces were probably in contact when the lips were closed.

CASE VI was a Russian who had been working in a lead-paint factory for but two weeks. He then was compelled to stop work and seek admission to the hospital on account of colic and constipation. There was much gingivitis with retraction of the gums and exposure of the encrusted roots. The blue line upon the gums was distinct about the insertions of all of the incisors and canines of both jaws, but it was not exceptionally dark or extensive. On the mucous membrane of the inner surface of both lips there was an area of blue discoloration, not removable by friction, and somewhat wider than was the line upon the gums. The discoloration was not uniform, as there were points of deeper color upon the slate blue discoloration of the whole patch.

CASE VII.—Peter McD., was an Irishman, aged sixty years. For five years he has been working in lead works as a laborer. After working for three months he had an attack of colic, for which he was admitted to hospital. Since that time he has had numerous milder attacks of colic which he has relieved unaided. He was again admitted to the hospital on November 30, 1894, for an attack of colic and obstinate constipation of three weeks' duration. The bowels had not moved for five days prior to admission.

His breath was very foul; the teeth encrusted with tartar, the gums retracted, and quite an active gingivitis was present at the margin of the gums. A distinct blue line was present on both gums, running well back beyond the canine teeth. On the inner surface of the upper lip directly corresponding with the line upon the upper gum, was a dark-blue area three inches in length and varying in depth from a quarter to three-eighths of an inch. There was no staining of the lower lip. The mucous membrane over the blue area had lost its smooth surface and was quite rough to the touch.

His colic was readily relieved by the ordinary methods of treatment. The blue coloration of the upper lip became distinctly less intense and less extensive during his stay in the hospital, but was still present at the time of his discharge, on January 2, 1895.

CASE VIII was an Irishman, aged thirty-three years, who was admitted to the Pennsylvania Hospital on January 14, 1895. He had been working in lead for three months. Three weeks before admission he was seized with an attack of colic, which continued until he sought relief at the hospital. The general condition of the mouth was fairly good. On both gums there was a distinct blue line near their margins. There was considerable retraction and inflammation of the gum around the left lower canine. Upon the mucous surface of the lower lip there was a long line of slate-blue discoloration in a position exactly corresponding with the gum margin in the lower jaw. A distinctly darker portion of this blue line corresponded exactly with the position of the inflammatory lesion in the gum about the left lower canine. The upper lip showed no blue stain. In other respects, the case did not differ from the ordinary run of cases of lead-colic, the intestinal condition readily yielding to treatment, and no other evidences of saturnism being present.

CASE IX.—Frank W., aged twenty-nine years, white, born in France, was admitted to the Philadelphia Hospital on May 3, 1895. He had been working in Wetherill's lead works for two years. The family history is negative in regard to gout.

About seven years ago he was injured in a mine explosion, the face being badly shattered and the skin deeply scalded. During the summer of 1894 he had an attack of colic with constipation, for which he was treated in the Presbyterian Hospital for five days. In September, 1894, soon after resuming work, he again had an attack of colic. Before the end of September he again started to work. He denies gonorrhœa and syphilis, and has been a moderate drinker of beer and porter.

From December, 1894, to the time of admission he had been suffering dull, aching pains, diffusely distributed, worse at sometimes than at others, and chiefly seated in abdomen and limbs. For two weeks before admission he had vomiting at intervals of several hours. This ceased two or three days before admission. Constipation was marked.

On admission he complained of severe cramp-like pain in the abdomen with diffuse aching pain in the extremities. He seemed stupid and listless. There was no sign of palsy, no nerve-trunk tenderness, no tremor or œdema. The face was much disfigured as a result of the mine explosion in which he participated seven years ago. The lips were much distorted and scarred, the lower jaw distorted and the teeth irregularly twisted out of relation with each other. On both gums there was a distinct deep blue line around the insertion of the teeth. This was most marked about the distorted teeth, where also there was marked gingivitis with retraction of the gum. The right lower lateral incisor was far removed from the neighboring canine, and between these teeth there was a deep fissure running downward into the jaw. At this point the lead staining was very marked. On the lower lip the inner surface was stained of a deep slate-blue color on a line corresponding to the gingival border of the lower jaw, while opposite the right lower lateral incisor and the right lower canine there was a flame-like area of intense discoloration exactly corresponding to the area of deeper staining mentioned as present between the two named teeth.

The urine was negative, save for a copious deposit of amorphous urates. After a few days' stay in the hospital the patient was discharged, cured of his attack. He was seen in June at the lead works, and had had no return of trouble. The condition of the mouth remained the same as at the time of his stay in hospital.

In connection with these cases the author wishes to record the results of his examination of some fifty workers in lead from points of view suggested by the above-related cases. The number of men examined is too small to permit of deductions being correctly drawn, but they are reported here as a small contribution to the statistical study of the phenomena of saturnism. For the privilege of examining the men the writer is indebted to Mr. W. H. Wetherill, who permitted the examination of the day force of laborers at his lead works. Dr. Henry Wetherill gave much assistance to the writer in his study of the men.

The day force of laborers was alone available for study. The pay-roll of laborers by day at the time of examination included about fifty men actually engaged in the handling of lead or its products, and excluding clerks, stablemen and others only indirectly brought in contact with the metal or its salts.

The chief products of the works are red and white lead, but the men are so transferred from one department to another that no continuous exposure to any one process of manufacture is possible. The works are all extremely well ventilated, and wherever it is possible mechanical means are substituted for manual in the handling of the metal, as in filling receptacles, sieving, and packing. To these circumstances, as well as to the careful instruction of the men in regard to their hygiene, is undoubtedly due the small percentage of men showing the effects of their exposure.

Forty-eight men were examined. Their ages ranged between eighteen and sixty-eight. Their nativity was as follows: Ireland, 27; United States, 13; Scotland, 3; Canada, France and Germany, of each, 1; not stated, 2. Twenty-two drank beer alone, two drank porter alone, one drank whiskey alone, seven drank beer and porter, three beer and whiskey, four beer, porter and whiskey, five were entirely temperate, and in three no statement is made in regard to alcoholism. Most of the men chewed tobacco to some extent, some to great excess. The time of exposure to lead varied from one week to twenty-six years. The average length of exposure among those working in lead for more than one year was nine and one-half years.

Of the forty-eight men, fifteen had had colic once, two twice, one thrice, while two had had many slight attacks of what was presumably lead colic. Three had had palsy of the extensors of the wrist, five had had weakness or stiffness of the hands, and two had had cramps in the thighs or legs. Three men suffered from headache, one from attacks of vertigo, one had had an attack consisting of a sudden convulsion followed by coma, and one had had an attack during which he was delirious for two weeks (supposed to be due to influenza, but possibly being a case of saturnine encephalopathy).

Arthralgic or myalgic symptoms had been present at some time in two of the men—one had much backache, one had pain with stiffness of the wrists at times. There were five others (mentioned above under the consideration of palsy) who had had at times stiffness of the wrists,

unassociated, however, with pain, and probably, therefore, representing slight grades of extensor paresis.

No history of gout was given by any of the men examined, and there were no evidences, in the form of articular, cutaneous or subcutaneous lesions, of such a condition, past or present, save in one case to be described below. The color of the mucous membranes was noted as follows: Good, fourteen; rather pale, two; slight pallor, sixteen; marked pallor, eleven; very marked pallor, two; not mentioned, two. The presence, absence and character of the lead line was observed in all but one of the cases. No line was present in nine who had worked in lead for the following periods: two weeks, four months, five months, two years, five years, nine years, ten years, fifteen years and sixteen years. In thirty-five the gums alone were involved, in two the lips also showed a blue discoloration (F. W., Case IX., and one other who had simply a small blue speck on the lower lip). Both gums were involved in nineteen (two not stated); the upper gum alone in three, the lower gum alone in eleven. The line was continuous in six, about the incisors in eleven, the incisors and canines in five, the incisors and molars in two. The line was described as narrow in seven, slight in sixteen, very faint in five, marked in two. Gingivitis was absent in thirty-one, slight in eight, marked in five; while in five there was marked retraction of the gum.

The influence of tobacco-chewing on the presence, absence or intensity of the blue line could not be positively determined, although the men with least blue line and gingivitis confessed to be excessive chewers of tobacco, and there was a firmly fixed idea among many of the men that the chewing of tobacco acted as a safeguard against the evil effects of lead. This idea may have some basis in fact, as it can be readily conceived that the man using tobacco in this way and expectorating at frequent intervals would be less apt to ingest lead particles, than would the man whose saliva and buccal mucus were swallowed. The absence of gingivitis in the chewers of tobacco may be due partly to the constant flushing of the mouth by the increased quantity of saliva formed by the act of chewing, partly also by the supposed antiseptic property of the saliva containing tobacco.

Extensor weakness was present in five of the men—in three both hands were involved; in two the right hand alone.

Tremor was present in nineteen; in twelve it was slight; in four

marked; in one the head alone was involved; in two it had existed since boyhood. In one of the marked and in four of the slight cases tobacco was used to excess. Owing to the fact that the men were examined while occupied in their more or less arduous labor, it was impossible to determine to what extent muscle-tire was responsible for the symptom, while, doubtless, to alcohol and tobacco was to be attributed some of the tremor.

Œdema was present in four cases, but in none to a marked extent. In two it was below the eyes; in two, of the feet. Nocturnal urination was not marked in any case, and was only present in six, in two of which it was only occasional and probably due to evening potations.

Pulse tension was very high in two; markedly increased in seventeen; slightly increased in four. The radial arteries were leathery in in four men, of which the ages were twenty-nine, thirty-four, thirty-seven and sixty-eight years. In the sixty-eight-year-old case, arcus senilis was very marked. The musculature was good except in the case of this same man.

No case of amblyopia directly traceable to lead impregnation was found. Constipation was present to any degree in but five men, and in them was not marked.

No signs of tophi in ears or eyelids were present, and there was but one case of articular gouty signs. This man was forty years of age, a drinker of beer and porter, who had been steadily employed for thirteen years in washing the crocks that were used in the tan-room, and in him the signs consisted of very marked bone proliferation about the articular ends of many of the bones of the hands. These thickenings were entirely quiescent, and were attributed by him to constantly repeated traumatisms in handling the crocks. He had no tophi or other skin lesions, and the only signs or symptoms, past or present, were pallor, a faint blue line on the gums, moderate gingivitis, and the articular changes in the hand.

It is, at first sight, remarkable that between two countries, having in some respects so many points of similarity, there should be so great a difference in the relative prevalence of gout as exists between England and America. As will be seen below, the hospital records of the two countries show a wide difference in the number of gouty subjects admitted to the wards, a fact that strikes everyone very forcibly in comparing the paucity of cases of gout in American hospitals with the

impressions created by the vast importance attached to gout in articles appearing in the British journals.

Among the better class of Americans gout is by no means uncommon, although less so than would appear to be the case in England. The reason for this comparative infrequency is not far to seek. The idle rich class in America is even now comparatively small, although, unfortunately, gradually increasing, and even this class has not as yet had time to suffer from the sins of gouty ancestors through the transmission of insufficient hepatic power. Among those who can be classed with the idle rich the majority are sons or grandsons of men far from wealthy. Inheriting quite good hepatic laboratories, it takes time for them to impair the efficiency of their hepatic functions, and many merely reach the stage of lithæmia without frank gouty outbursts. The lithæmic state is far from infrequent in America, doubtless, in part, on account of the active nervous strain felt more or less by all in the struggle, not for existence, but for the attainment of a better position in the community wherein the high places are attainable with marvelous rapidity. Moderation in meat and drink, with continuous nervous tension, may explain the relative infrequency of gout and frequency of lithæmia in more or less pronounced degree among Americans of native parentage.

The hospital class in Philadelphia may be said to be composed of native Americans, Irish, Italians, Russians, Germans, English, Scandinavians, Scotch and Austrians. As an illustration of the composition of the hospital population in Philadelphia the statistics of the Pennsylvania Hospital may be taken. Between the years 1886 and 1895 there were admitted to the wards 22,345 patients. Of these, 10,396 were natives of the United States, 4351 of Ireland, 1216 of Russia and 6382 of other nativity. During the year just completed, 2375 patients were admitted to the wards. The nativity of these was distributed as follows: United States, 1069; Ireland, 297; Italy, 246; Russia, 234; Germany, 164; England, 107; Sweden and Norway, 43; Scotland, 36; Austria, 31; Poland, 25; France, 13; Hungaria, 11.

As an aid toward accounting for the rarity of gout in American hospitals as compared with those in England, a brief glance at the conditions of life among those who seek admission thereto may be of some value. The Italians, Russians, Poles and Huns are in Philadelphia among the most degraded of any peoples coming from civilized

countries, and include either the most poverty-stricken classes of their native countries, who live in a state of poverty and filth in their new home, or of the laboring classes of their native countries to whom the relatively high wages and cheap living of America have offered inducements to emigrate temporarily for the sake of returning home with money, not only earned by their labor but saved by their economical and highly unhygienic manner of living in the place of their temporary adoption. Among such as have been mentioned even "poor-man's gout" can hardly be conceived of. A sufficiency of food is obtainable by all, their habits in regard to alcohol are, on the whole, temperate on account of their eager pursuit of money, and the work of most of them (with the exception of the Russians) is sufficiently active to keep elimination up to a standard that precludes any accumulation of waste material within their economies. Natives of the British Islands, Germany, Scandinavia, Austria and France are, as a rule, hard-working, temperate and fairly well-to-do people, who lead a quite hygienic life with active labor, a sufficiency of wholesome food, good clothing, and a fair amount of enjoyment of life. Of the native Americans the same can be said as of the latter class. Beer and whiskey being the favorite beverages, and comparatively little porter or wine being consumed, may, in part, account for the small amount of gout seen in this part of the world. In regard, however, to alcohol as the agent responsible for the production of gout, it has always seemed to the writer that its action might be aptly compared to that of medicinal doses of mercurials; given in large amount at once, calomel, for instance, purges and has, so to speak, a local action; given continuously in divided doses it salivates. As one of the characteristics of American alcoholics is the indulging in a periodical "spree," followed necessarily by a hard day's labor to "work it off" and so get rid of extra waste products, the comparison to a large dose of mercurial is not unjustifiable. When an American of the lower classes is a constant drinker, the alcohol is usually taken in the form of whiskey, which seems to have little power of predisposing to or exciting an attack of gout.

This comparative indemnity from gout is, however, not a peculiarity of America alone. This is well shown by the facts given by Hirsch in his "Hand-book of Geographical and Historical Pathology," but is even better attested by the statements quoted below.

The answers to Sir Dyce Duckworth's[1] letters of inquiry to physicians throughout Great Britain show some interesting facts. Drummond wrote him from Newcastle-on-Tyne that gout was rare in that place, and was never seen among the lower classes; that lead poisoning was common, but was never associated with gout. Of Glasgow, Gairdner wrote that both gout and saturnism were rare in that city, and that he never saw a case of lead impregnation in association with gout having its genesis in Scotland. Sir Walter Foster wrote Duckworth, from Birmingham, that plumbism was not common there, and that there was but little gout among the lower orders. The reply from Dr. Wynne Foot states that in Dublin saturnine arthritis is familiar; while Professor Cunning wrote from Belfast that he saw no connection between gout and plumbism. In Liverpool, according to Duckworth, both gout and plumbism are rare. Sir Robert Christison wrote to Garrod[2] that in Edinburgh both plumbism and gout were rare. In response to a request from Sir Dyce Duckworth, Frerich analyzed one hundred and sixty-three cases of chronic lead poisoning without finding a case of gout, and states that gout is rare in Berlin. Duckworth himself gives the results of the examination of one hundred and thirty-six cases of gout in hospital practice, and found that 18 per cent. showed signs of lead poisoning and worked in that metal. Garrod found that at least one out of every four gouty patients in hospital had been plumbers or painters. Richardiére[3] says of Paris that saturnine gout is very rare in that city.

It is seen, therefore, that, as a rule, the prevalence of ordinary gout and of that produced by lead intoxication together varies very much in different localities.

It has been shown by Garrod that lead poisoning and gout stand in close relation to each other—the gouty readily becoming intoxicated with lead, the lead-worker being a ready subject for gout. In America, therefore, where gout is relatively infrequent, we should expect to find less plumbism than, for instance, in England. In America, or at least in Philadelphia, plumbism is by no means rare, showing itself most commonly as wrist drop or colic, less commonly as encephalopathies, still less as arthralgias, and with extreme rarity as

[1] Sir Dyce Duckworth "A Treatise on Gout," 1889, p. 156.
[2] A. B. Garrod "A Treatise on Gout and Rheumatism," 1876, p. 236.
[3] Richardiére, Traité de Médicine, Vol. II.

gout. Gout from other causes is, as has been said, extremely rare in the Philadelphia hospitals, as will be seen at a glance in the following table, so arranged that the relative number of cases of gout and the different forms of lead poisoning may be compared with those admitted to Pennsylvania, St. Bartholomew's and Middlesex Hospitals during a corresponding period.

	Pennsylvania (including Out-patient Dep't).	St. Bartholomew's.	Middlesex.
Total of medical cases treated in ten years	30,460	23,684	13,274
Cases of gout	*47	167	132
Cases of plumbism	189	131	50
Percentage of gout to total admissions	0.1543	0.8580	0.9954
Percentage of plumbism to total admissions	0.6240	0.4897	0.3766

It will be seen by this table that the number of cases of lead poisoning, as compared with the total number of medical cases treated, is far greater in the Pennsylvania than in the other hospitals, while in the latter the percentage of gouty patients is far in excess of those at the Pennsylvania Hospital, even when with gout are included the cases of allied conditions that were treated at the hospital here.

*Including "uric acid gravel" and "uric acid diathesis."

TREATMENT OF RHEUMATISM WITH THE SALICYL COMPOUNDS.—AN HISTORICAL NOTE.

By FREDERICK P. HENRY, M.D.

A REMINISCENCE, in order to be historical, should be connected with an important fact of more or less ancient date. If the fact is of universal interest, so much the more does it merit the epithet "historical." Its age is a subordinate matter, although the lapse of time may invest it with additional dignity. Some facts are epoch-making; *i. e.*, open a new era and so take their places as historical by birthright, while others are slowly, even grudgingly, accepted, and continue to maintain a precarious position.

The introduction of the salicyl compounds into the therapeutics of rheumatism was certainly epoch-making, and since they have maintained their position for nearly twenty years, it has seemed to the writer appropriate that he should publish his personal knowledge of their history.

The salicyl compounds employed in the treatment of rheumatism are three in number: (1) salicylic acid and its salts; (2) salicin; (3) methyl salicylate or oil of wintergreen. The attention of the profession was first called to the success of the first-mentioned drug in the treatment of rheumatism by Dr. Stricker, of Berlin, in 1876. In January and succeeding months of 1877 I administered salicylic acid to a large number of rheumatic cases at the Episcopal Hospital of Philadelphia, and, so far as I know, this was the first hospital in this city, if not in this country, in which the drug was employed on a large scale.

When a student at the College of Physicians and Surgeons of New York, I had heard the late Prof. Alonzo Clark remark that, in his opinion, the greatest therapeutic advance of the day was to be found in the alkaline treatment of rheumatism introduced by Dr. Fuller, of London. I soon had convincing reasons to believe that the alkaline treatment was eclipsed.

I do not claim the sole credit of introducing this treatment into the Episcopal Hospital, where, it is needless to say, it has been employed ever since. The suggestion of its use was made by my then resident physician, the late Dr. Charles B. Goldsborough, formerly surgeon of the Marine Hospital service, and I am glad of the opportunity of paying this tribute to the memory of my former pupil and friend. Dr. Goldsborough, a graduate of the University of Pennsylvania, and a pupil of the late Prof. Carson, selected salicylic acid as the subject of his graduating thesis, this acid having, about that time, attracted attention on account of its decided antiseptic properties. Dr. Goldsborough's thesis was awarded the first prize, or, to be strictly accurate, the first prize was divided between him and Dr. Robert Meade Smith, the theses of these two gentlemen being considered of equal merit.

While acting as resident physician at the Episcopal Hospital, Dr. Goldsborough, whose interest in salicylic acid continued, saw some reference to the marked success which had followed its administration in a number of rheumatic cases in Germany, and asked whether I would be willing to employ it in the wards under my care. It is needless to say that I consented, for I had seen enough of rheumatism to feel convinced that its treatment was one of the opprobria of medical practice. Suffice it to say that the success which followed the use of the new drug afforded a wonderful contrast to the results which had preceded it.

During our first employment of salicylic acid at the Episcopal Hospital, we administered the drug in capsules, or rather in *cachets* (*cachets de pain*). Soon after it was found that the soluble sodium salt was equally efficacious, and the administration of the pure acid fell into comparative disuse. Of late there has been a tendency to return to the latter, and the writer regards this retrograde step as a wise one, for, in his opinion, the *pure acid* is more effective than any of its compounds.

It is eighteen years since the treatment of rheumatism by salicylic acid and its sodium salt was first employed in Philadelphia. During that period numerous drugs have been highly recommended for various diseases, have had their brief period of so-called success and been forgotten, but when salicyclic acid was introduced it came to stay.

A fact of somewhat amusing interest in connection with this subject is that the virtues of salicylic acid are questioned or denied, as a rule, solely by those who have not practiced in pre-salicylic days.

To Dr. Maclagan, of Scotland, belongs the credit of having first employed one of the salicyl compounds in the treatment of rheumatism. The substance used by him was salicin, and he was led to employ it on purely theoretical grounds. Believing in the miasmatic origin of rheumatism, he sought for a remedy among the plants and trees which grow in malarial districts, and found it in the willow. He began to use salicin, a bitter principle found in the willow, in 1874, and published his results in 1876. It has since transpired, although the fact was unknown to Maclagan, that the Dutch Boers and native Hottentots have long been accustomed to use a decoction of willow shoots as a remedy for acute articular rheumatism.

Although this note was intended to be purely historical, it may not be amiss to refer to the present status of the salicyl compounds. In 1892 Dr. Marcel Baudouin[1] made a tour of the Paris hospitals with a view to ascertaining the prevalent treatment of acute articular rheumatism in those institutions. He obtained reports from Professors Straus and Bouchard, MM. Millard, Guyot, Dujardin-Beaumetz, Bucquon, Talamon, Chauffard, Barth, Barié, Comby, Faisans, Sevestre, Ollivier and Simon. The verdict is overwhelmingly in favor of salicylate of sodium. Straus, Dujardin-Beaumetz and Faisans regard it as a veritable specific; Millard, as the remedy *par excellence*. Guyot speaks of the introduction of this drug into the treatment of rheumatism as a therapeutic[2] triumph, and the same opinion, practically, is expressed by all the clinicians mentioned. In the treatment of these practitioners there are minor differences as to dose and mode of administration, but the chief reliance of all is upon the drug in question.

[1] La Semaine Médicale, 11 Mai, 1892.
[2] L'une des rares conquêtes de la thérapeutique actuelle.

NOTE ON THE USE OF ACETANILID AND RESORCIN IN THE LATER STAGES OF PHTHISIS.

By SAMUEL WOLFE, A.M., M.D.

During my term of service in 1893 the phthisis ward contained a very large number of cases in the advanced stages of the disease. Dr. Davis was acting as my resident, and, largely at his suggestion, we gave to a considerable number of cases five-grain doses of acetanilid at intervals of three or four hours, with a view of relieving within a degree, the distress which so many of these patients suffer. It was noted that under this influence the daily range of temperature was less, cough and diarrhœa were less troublesome, and the patients ate and slept somewhat better. Antipyrin, phenacetin, exalgin and kindred preparations were not tried. Such a trial might result in showing one or the other of them superior to acetanilid for this purpose.

Resorcin I gave in three and four grain-doses, at intervals of four hours, for the control of diarrhœa, and found it effective. It seemed, besides, to exert a slightly favorable influence on the temperature. It would seem to me that these agents might deserve a fuller trial. The question of affording comfort to advanced tubercular cases is certainly very often a puzzling one for the practitioner, and any agents of service in this field that are without any specially objectionable features should have assured them a free trial, and, on proof of worth, a grateful reception.

A STATISTICAL STUDY OF INFLUENZA; ITS POTENCY TO LESSEN THE RECEPTIVITY OF THE BODY TO MALARIA, AS WELL AS TO INCREASE THE RECEPTIVITY TO PNEUMONIA AND PROBABLY TO TYPHOID FEVER.

By J. M. ANDERS, M.D., Ph.D.

The application of improved methods of bacteriological research, in recent times, has resulted in the enlargement of the class of diseases known as the acute and chronic infectious diseases. In this paper it is not intended to discuss the disease-producing bacteria, but the differences in the behavior of the latter, due to modifications of the tissue-soil, in consequence of the occurrence of other diseases belonging to the same category.

The conditions upon which the growth and development of the specific bacteria depend (after they have invaded the human system), are various, and, many of them, it must be confessed, are, as yet, wholly misunderstood.

Among known, and generally accepted external influences, are *chemical characters of the soil and water*, the presence or absence of certain mineral constituents in the *nutrient media*, which are used for the culture and growth of the bacilli (particularly acids and alkalies); and the presence or absence of free oxygen, anaërobic microphytes (which infest the human system) flourishing best without this agent. But we do not know the determining factors when, for instance, two persons are equally exposed to a specific contagion, and the one becomes infected, while the other escapes the disease. These differences in the susceptibility of the tissues are inexplicable. It may, however, be assumed safely that the varying degrees of receptivity of the human organism must arise from minute variations in the chemical constitution of the liquids and solids of the body. But the precise character of these changes is unknown. The operation of certain causes may

increase individual susceptibility. For example, *age* (owing to changes in the composition of the liquids of the body at different life-periods), exposure to extreme cold or heat, ordinary "cold" or other slight ailment, may each induce sufficient disorder of the bodily functions to alter slightly the liquids of the body, and, thus, invite bacterial invasion.

In like manner infection with a specific bacillus may be favored by the recent occurrence of an infectious disease. We have instances of this in measles and typhoid fever, which are sometimes followed immediately by pulmonary tuberculosis. *Per contra*, the occurrence of an infective disease may not only establish immunity from the disease itself, as in the case of measles, scarlatina, etc., but also lessen, or even destroy, the receptivity to certain other diseases of the same class.

Unquestionably, our knowledge pertaining to the influence of the occurrence of acute infective diseases in predisposing the system to others of the same class on the one hand, or in diminishing the susceptibility of the system to these affections on the other hand, is as yet quite limited. We are equally ignorant of the exact duration of periods of increased or diminished receptivity to infective diseases brought about by their occurrence. Believing that any new facts which would throw additional light on these unsolved problems would be welcomed by the profession, the writer has made a collective investigation into certain acute, infectious diseases, particularly with reference to the points at issue.

The chief object of the present paper, however, is to illustrate by statistical evidence: (1) that influenza lessens the receptivity of the body to malarial infection, and (2) influenza increases the bodily susceptibility to typhoid fever (?) and pneumonia. Human susceptibility to the bacillus of Pfeifer, which is the specific cause of influenza, is more general than in the case of any other acute infectious disease, if we except variola. But, whilst during a pandemic outbreak of la grippe none are exempt, yet its development is, to some extent, under the influence of the conditions that modify the liability of the body to other infective diseases as a class, especially *age*. Thus, very young children are less receptive to this particular poison than older individuals, though the latter circumstance may be due in part to the fact that the very young are less exposed to the contagion than older subjects. A primary attack of influenza does not immunize the body,

since quite frequently before convalescence is completed, a relapse speedily develops. Not only so, but the third or even fourth successive attack may occur, though rarely.

(1) *Epidemic influenza diminishes the receptivity of the body to malaria.*—The cases analyzed were furnished by the records of the medical wards—both male and female—of the Philadelphia Hospital. For reasons which will appear obvious hereafter, the period of time covered by these observations—eight and one-half years—falls conveniently into two divisions, the first being three and one-half years (from January 1, 1886, to July 1, 1889), and the second five years (from July 1, 1889, to July 1, 1894).

In this connection I desire to make grateful acknowledgment of the valuable services rendered by Dr. A. M. Davis in the preparation and examination of the records from which the statistics were gleaned. My thanks are also due Drs. Robert Yenney and Wm. F. Kleinstuber for kind assistance in the collection of data from the books of the hospital.

The following simple table shows the total number of individual records which have been investigated, and the number of patients suffering from each of the above-mentioned diseases.

TABLE I.[1]

	Influenza.	Malaria.	Typhoid.	Pneumonia.
Jan. 1, 1886, to July 1, 1889 . . .	1	364	208	284
July 1, 1889, to July 1, 1894 . . .	337	247	272	346

It will be seen from a glance at the table that for the three and one-half years, or first division of time, the number of cases of malaria was relatively much greater than for the five years, or second division, although the sum-total of patients admitted into the medical wards from year to year varied but little. It may be pointed out, in this connection, that after the cases of influenza had for some months practically ceased to be admitted in 1894, patients suffering from malaria began to seek admission in a proportionately greater percentage than during the period of five years preceding, or when influenza was prevalent. Thus, for the months of July, August and September, 1894, there were but two cases of influenza and thirty-six (36) of malaria.

[1] The date of admission was taken as a basis from which to reckon the number of cases.

Facts such as these strongly suggest the thought that influenza is potent to lessen the receptivity to malarial infection. It is to be recollected, however, that highly malarial localities may in time become free from the disease as the result of the operation of certain causes, such as exposure of the surface soil to the influence of the sun-light, its prolonged cultivation, etc. But the latter view is scarcely tenable as relating to the patients that are admitted into the Philadelphia Hospital, since they come from various and widely-separated districts of the city, more or less malarious.[1] That influenza tends to destroy the receptivity of the body to the organism of Laveran is also shown by the subjoined table.

TABLE II.

	Influenza.	Typhoid.	Malaria.
April 3, 1891, to May 4, 1891	50	11	2
April 3, 1888, to May 4, 1888	0	9	16

An inspection of Table II. will show that only two cases of malaria occurred during the time in which fifty cases of influenza occurred. Moreover this ratio obtained at a season of the year when cases of malaria are not infrequent at the Philadelphia Hospital, as shown by the number—sixteen—admitted in the corresponding period of the year 1888. The latter year was selected for comparison, owing to the fact that it immediately preceded the first among the more recent epidemic outbreaks of influenza. Other previous years furnished an equally large number of cases of malaria.

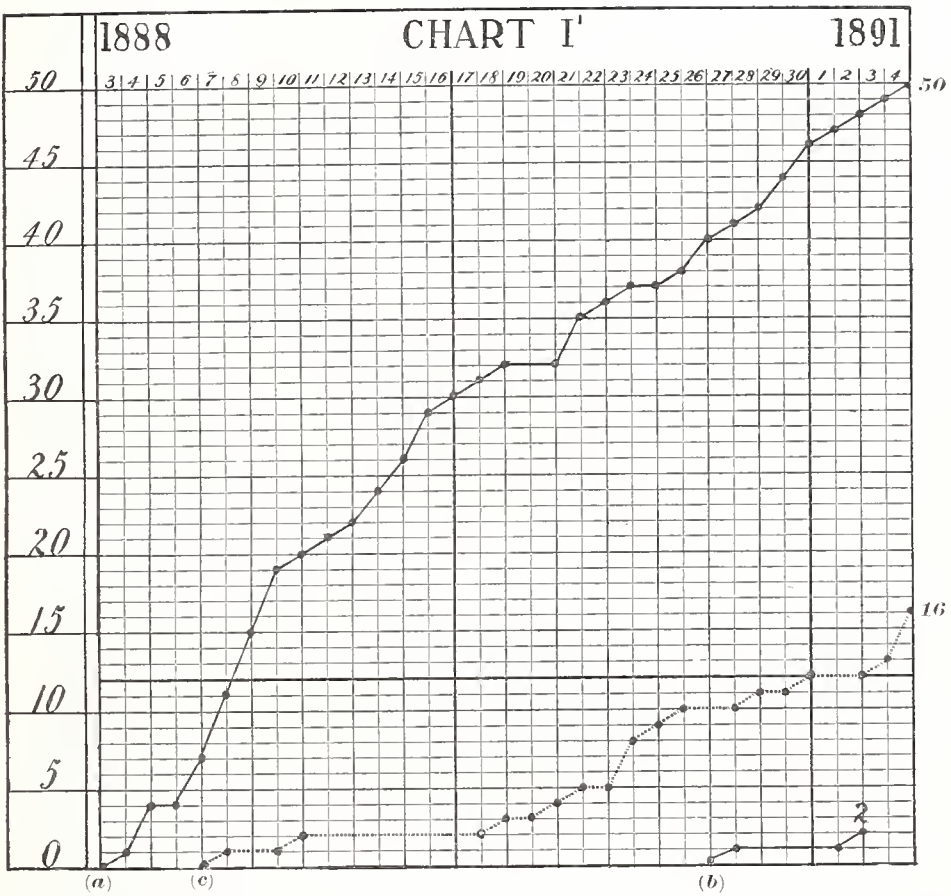
Again, the two cases of malaria which occurred during the time embraced in the table were admitted near to the close of the epidemic period, or on April 27, 1891, and May 2, 1891, when influenza cases were rapidly diminishing in frequency.

The tracings in Chart I., which were based upon the data contained in Table II., also illustrate the effect of influenza upon the appearance of malaria.

A comparison between the number of cases of malaria, typhoid fever and pneumonia that were admitted during the first recent influenza epidemic which prevailed in the Philadelphia Hospital (December 26, 1889, to January 28, 1890, inclusive), and the number of cases of

[1] It must be confessed that the proportion of the total number of cases of malaria that were admitted from other departments of the hospital into the medical wards has not been ascertained.

these affections for a corresponding period of the year, when no influenza prevailed, was also attempted. But it was found that the cases of malaria during the months of December and January, in



Traciug (*a*) represents the number of cases of influenza for each day, as well as the total number of cases occurring during the period of the spring epidemic (April 3, 1891, to May 4, 1891, iuclusive). Tracing (*b*) represents the total uumber of cases of malaria for the epidemic period, and also shows the days on which the cases appeared. Traciug (*c*) represents the total number of cases of malaria which occurred during the corresponding period of another year (April 3, 1888, to May 4, 1888, inclusive), when there were no influenza cases, and the days ou which the various cases occurred.

seasons when no influenza prevailed, was so small as to render any data thus obtained unavailable for the purpose intended.

During the period of five years, from July 1, 1889, to July 1, 1894, in which there occurred 247 cases of malaria and 337 cases of influenza,

[1] Comparison of the number of cases of malaria which occurred during the period when influeuza was epidemic (April 3, 1891, to May 4, 1891, inclusive), with the number of cases of malaria which occurred during a correspouding period of another year (April 3, 1888, to May 4, 1888, inclusive), when there were no cases of iufluenza.

only six of the former gave a history of the latter. It is interesting to note that in one of these latter instances the malaria developed one month after the attack of influenza; in another, seven months after; in two, the interval was one year; and in the remaining two it was not less than four years. Hence, if the immunity bestowed by the occurrence of influenza should not be permanent, and we may reasonably presume that it is not, then the instances in which temporary immunity, at least, is not given by an attack of influenza, would appear to be rare indeed, according to these observations. The paucity of cases in which a prior attack of la grippe occurred in the numerous malarial subjects, at a time when the community from which those admitted came passed through several epidemics of the former disease, is, to say the least, highly significant, and serves to strengthen the basis for the inference that influenza tends to destroy susceptibility to malarial infection. But there are still other corroborative circumstances.

Thus, out of 247 cases of malaria admitted into the institution during the five years or second period of time, in 86 there was a previous history of malaria, and 43, or 50 per cent., of those who furnished the said history had malaria within the period before mentioned, or during the time of prevalence of influenza. These data are also confirmatory of the well-known fact (*supra*) that a primary attack of malaria predisposes to future attacks. It should be added that in 30 of the cases of genuine malaria there was an antecedent history of typhoid fever, and that 12 of the latter cases took place during the five-year period in which influenza prevailed. Obviously, the prophylactic immunity given by influenza, is not observed in the case of typhoid from malaria fever. It is of considerable interest to note that a history of prior pneumonia was obtained in but 9 cases, 4 of which occurred in the five-year period; *neither was pneumonia noted as a complication of malaria in a single instance*. It is therefore highly probable that so-called malarial-pneumonia is rare in the vicinity of Philadelphia. During the time of the epidemics of influenza the general mortality-rate for Philadelphia was doubled in consequence of the increased prevalence of fatal forms of illness; and yet few cases of malaria were treated at the institution during those brief periods.

When the occurrence of one acute infectious disease leaves the system immunized from the infection with certain other specific microorganisms, the system may be said to be vaccinated by the ptomaines

of the primary affection. As to the duration of the immunity against malaria that is conferred by influenza, we know as yet nothing definitely.

I am persuaded, as the result of more extended observation along this line, that important advances in medical science will be made in the direction of artificial protective inoculation. Serious affections may in this way become preventable by the use, not only of their own pathological bacilli, as in the cases of tetanus and diphtheria (?), but also by the use of bacilli of other affections which are either benign or infinitely less dangerous to life. Except for the circumstance that the serum of the blood of an animal that is rendered immune by the pathogenic organism of another disease is employed, the basis of the treatment suggested by these investigations is the same as the Behring serum treatment of diphtheria, or the serum treatment of tetanus. Obviously, the fact that in pursuing the application of this method the system may be rendered more susceptible to other forms of bacterial infection, needs to be carefully weighed. Since immunity given by serum is "passive," according to Ehrlich (that is, lasts only while the serum is circulating in the blood), I suggest that it may be found (upon further experimentation) that the Pasteurian method of immunizing with attenuated virus may be practiced where the poison of one infectious disease affords protection against another of the same class. This latter course would result in so-called "active" immunity, which is supposed to be more or less permanent, since the use of attenuated virus modifies the tissue in a manner similar to the effects of the disease themselves. As a practical illustration of this mode of prophylaxis, the immunization of the body from small-pox by means of vaccination with cow-pox may be cited. In instances of this sort, a true alterative effect is produced upon the tissue soil by the protective virus. Naturally, prolonged immunity may also be secured by using the serum, if the injections are repeated at sufficiently short intervals.

Although what I shall term the "Substitution" method of imparting immunity may in future amount to little in practice, a knowledge of the fact that the specific virus of one infectious disease produces immunization of the body from others is of real scientific value and of practical advantage to the profession.

(2) *Influenza increases the bodily susceptibility to typhoid fever and*

pneumonia.—The opposite effect of influenza is also shown by these observations. Thus, during the time of the *epidemic appearance of influenza* an increased prevalence of typhoid probably occurred. Table I. shows that there were 208 cases of typhoid fever during the first period of three and one-half years, and 272 cases during the second period of five years. These figures indicate a decrease of about 8½ per cent. for the years in which influenza prevailed. But when we consider only those comparatively brief periods of time during which influenza was truly epidemic, it will be seen hereafter that there was a slight, though decisive, increase in the cases of typhoid fever. I had observed during the early epidemics of influenza which occurred since the autumn of 1889, several cases that appeared to be typical influenza merge into true typhoid fever.

In the autumn of 1893, two instances of this sort fell under my care:

CASE I. occurred in a male, aged twenty-two; occupation, commercial traveler. I was called October 28, 1893, when I found him suffering from all the characteristic symptoms of la grippe. The case began with a rigor, accompanied by high fever, intense headache and pains in the back, in the eyes, and the muscles generally. Marked constitutional depression was present from the time of onset. After a couple of days had elapsed, local symptoms appeared. These were coryza, hoarseness, and later severe paroxysms of cough. As the "dry" bronchitis began to subside, gastro-intestinal disturbances, as shown by nausea, occasional vomiting and diarrhœa, appeared. The gastric symptoms were attributed to influenza, and, after lasting a few days, they subsided. The fever, however, did not abate, but continued to range from 102⅘° F. to 104° F.; diarrhœa of moderate grade (the stools being of bright yellow color) also continued; the spleen was now found to be enlarged. There were no marked nervous symptoms and no marked complications. The characteristic typhoid spots put in an appearance on the twelfth day of the affection, manifesting themselves in three successive crops. The fever declined by lysis, the temperature touching the normal on the twenty-eighth day of the disease. At the end of an afebrile period lasting one week, a true relapse occurred. The duration of the latter was two weeks. The patient recovered.

Though a bacteriological study of the bronchial secretion was not made in the above case, the history and general and local symptoms, surely seemed to justify the diagnosis of "la grippe," and the case was regarded as such during the first few days of the attack. Of the fact that it was followed promptly by typhoid there can also be no doubt.

Unfortunately, the notes of the second case have been mislaid, but since it was of comparatively recent occurrence, and since it was

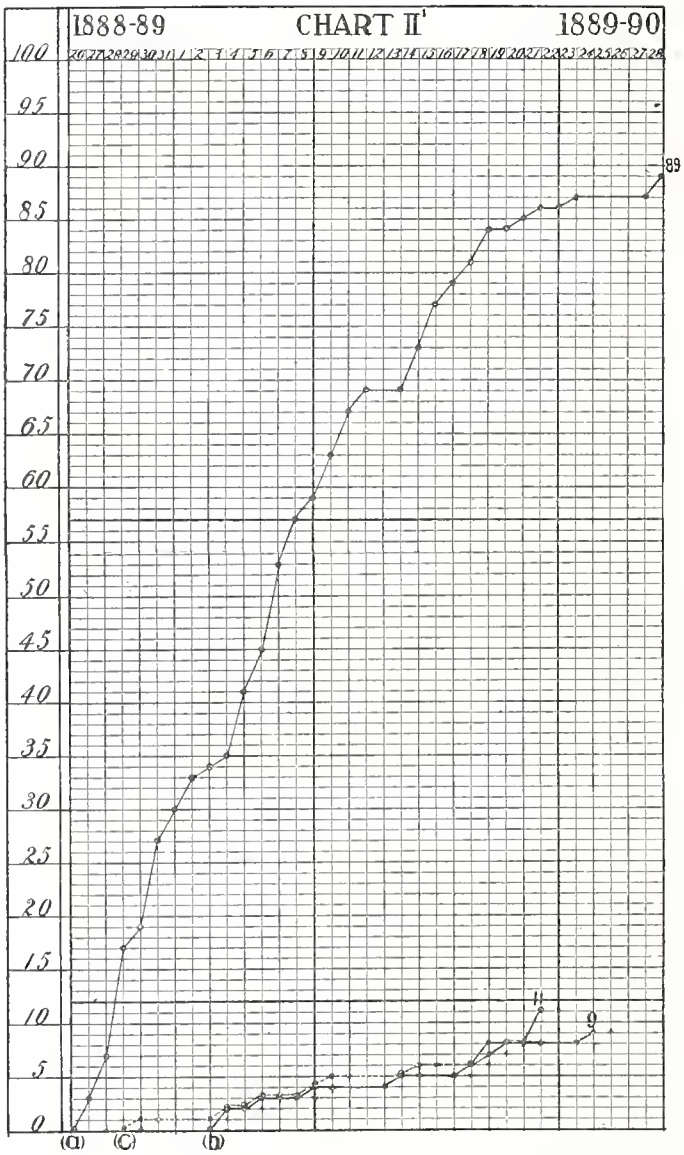
used repeatedly for ward-class teaching, the leading particulars are easily recalled:

CASE II.—The subject was a male, aged about forty years, who was admitted into the medical wards of the Philadelphia Hospital in the latter part of September or early part of October, 1893. He came presenting all of the clinical features of influenza which need not be related here. After treatment for this affection had been carried out for a week or ten days, the usual diagnostic symptoms of typhoid appeared and soon overshadowed those of the primary disease. The spleen became decidedly swollen; the temperature, which became quite elevated, pursued a continued type (defervescence by lysis). The typhoid spots which later came out on the trunk, served to place the diagnosis beyond peradventure. So prominent were the local symptoms (referable to the bronchi) in this instance, as to arouse suspicion of phthisis. The absence of bacilli as demonstrated by repeated examination of the sputum, and the absence of the characteristic physical signs, however, enabled me to exclude pulmonary tuberculosis. The case terminated in complete recovery.

The records of the Philadelphia Hospital which furnished in the aggregate 338 cases of influenza, also support the view that the latter complaint leaves the body in a condition of heightened receptivity to typhoid. In the first place typhoid fever was found to be noted as a complication in seven cases of influenza. In one of the latter, it "preceded the typhoid;" in another, the temperature had been normal for twenty days, when typhoid fever developed. The epidemic influence of la grippe on the prevalence of typhoid fever is better shown by the two following charts.

A careful study of the tracings in Charts II. and III. will convince the most skeptical that the number of typhoid sufferers increased with the decided prevalence of influenza, though the increase cannot be said to have been marked. It is conceded that to show the constancy of this ratio, more extended researches of a similar character must be undertaken. The facts are detailed for what they are worth to indicate the probable etiological connection between two important, acute, infectious diseases. It is not claimed here that all modifying conditions and circumstances have been eliminated, but this has been, so far as possible, attempted.

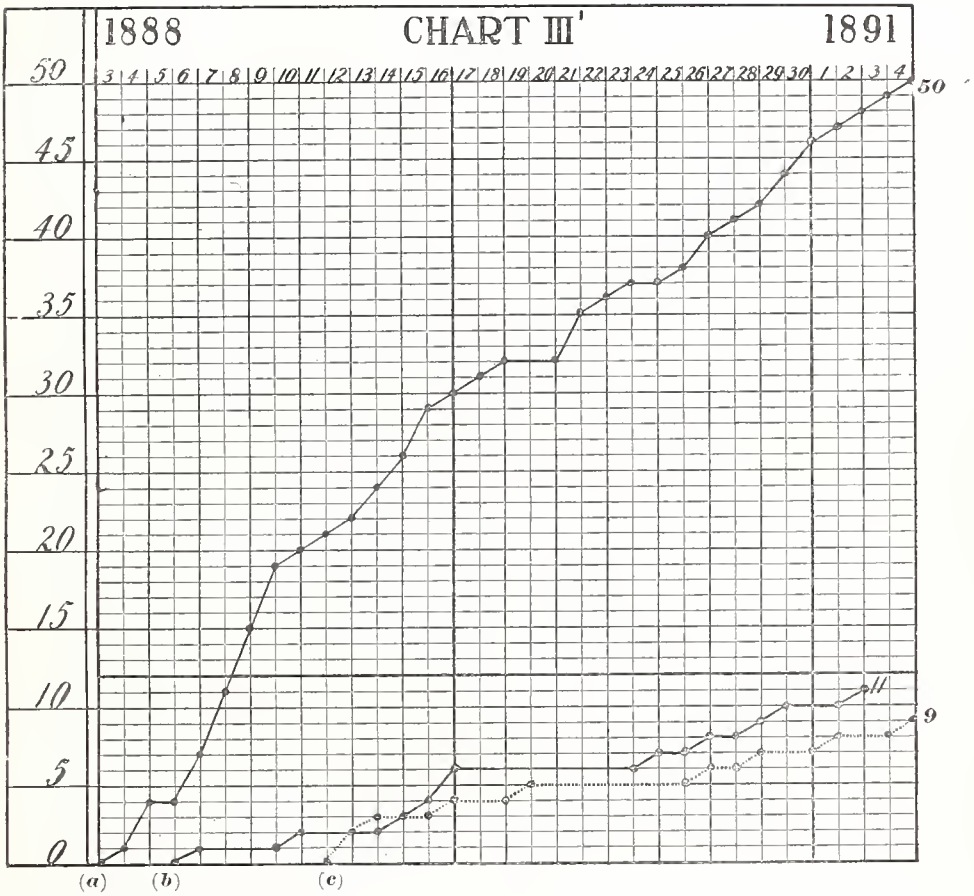
That the bodily receptivity to pneumonia is heightened by the prevalence of influenza is clearly demonstrated by the present researches. Thus, if a comparison of cases for the whole period of five years (446), during which influenza prevailed from time to time, with the number which occurred during the previous three and one-half years (284) is drawn, it will be found that the ratio of cases for the former



Tracing (*a*) represents the number of cases of influenza for each day, as well as the total number of cases occurring during the period of the winter epidemic (December 26, 1889, to January 28, 1890, inclusive). Tracing (*b*) represents the total number of cases of typhoid fever for the epidemic period, and, also, shows the days on which the cases occurred. Tracing (*c*) represents the total number of cases of typhoid fever which occurred during the corresponding period of the previous year (December 26, 1888, to January 28, 1889, inclusive), when there were no influenza cases, and the days on which the various cases occurred.

[1] Comparison of the number of cases of typhoid fever which occurred during the winter epidemic of influenza (December 26, 1889, to January 28, 1890, inclusive), with the number of cases that occurred during the corresponding period of the previous year (December 26, 1888, to January 28, 1889, inclusive), when there were no cases of influenza.

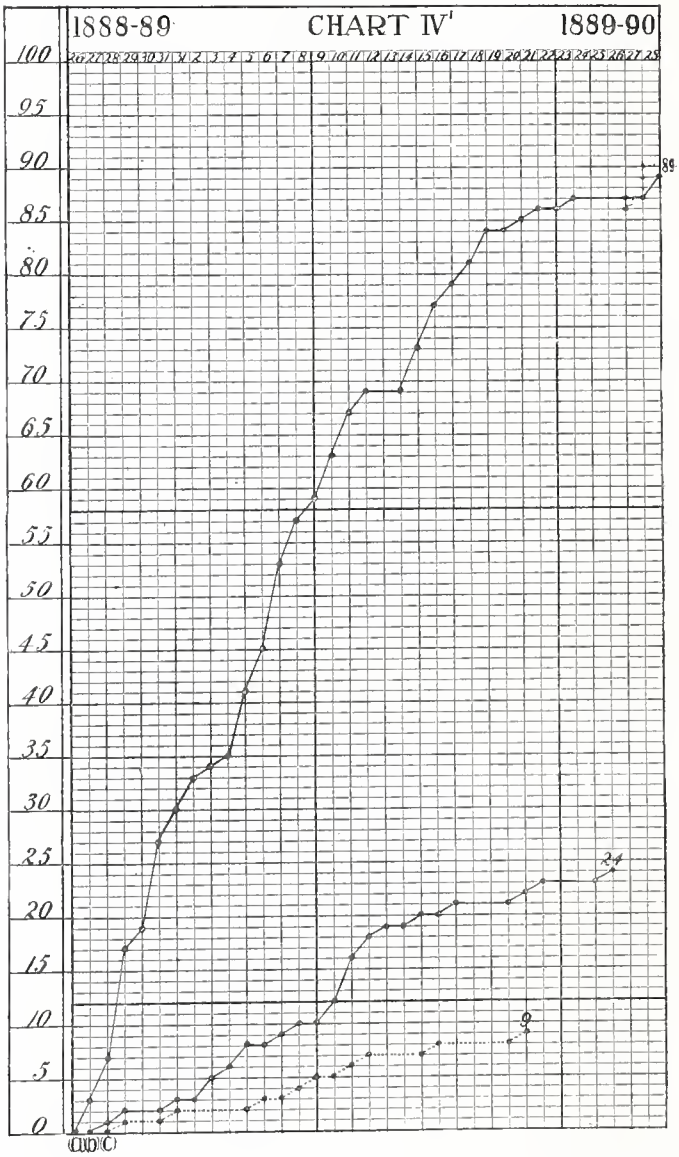
interval is in excess of that for the latter. This augmentation of cases of pneumonia, doubtless, in part, the result of prevalent influenza, becomes striking when definite lengths of time, during which influ-



Tracing (*a*) represents the number of cases of influenza for each day, as well as the total number of cases occurring during the period of the spring epidemic (April 3, 1891, to May 4, 1891, inclusive). Tracing (*b*) represents the total number of cases of typhoid fever for the epidemic period, and shows the daily occurrence of the cases. Tracing (*c*) represents the total number of cases of typhoid fever which occurred during the corresponding period of a prior year (April 3, 1888, to May 4, 1888, inclusive), when there were no cases of influenza, and the days on which the various cases occurred.

enza prevailed extensively, are considered. So far as the influence of influenza goes in inviting secondary disease of the lungs, particularly catarrhal and lobar pneumonia, these investigations do not show anything that is new, but strongly corroborate the known results of pre-

[1] Comparison of the number of cases of typhoid fever which occurred during the spring epidemic of influenza (April 3, 1891, to May 4, 1891, inclusive), with the number of cases that occurred during the corresponding period of a prior year (April 3, 1888, to May 4, 1888, inclusive), when there were no cases of influenza.



Tracing (a) represents the number of cases of influenza for each day, as well as the total number of cases occurring during the period of the winter epidemic (December 26, 1889, to January 28, 1890, inclusive). Tracing (b) represents the total number of cases of pneumonia for the epidemic period, and, also, shows the days on which the cases occurred. Tracing (c) represents the total number of cases of pneumonia which occurred during the corresponding period of the previous year (December 26, 1888, to January 28, 1889, inclusive), when there were no influenza cases, and the days on which the various cases occurred.

[1] Comparison of the number of cases of pneumonia which occurred during the winter epidemic of influenza (December 26, 1889, to January 28, 1890, inclusive), with the number of cases that occurred during the corresponding period of the previous year (December 26, 1888, to January 28, 1889, inclusive), when there were no cases of influenza.

vious observations relative to the subject. There occurred during the winter epidemic of 1889 and 1890 (*supra*) twenty-four (24) cases of pneumonia, as against an average of 12.6 cases for the same period in the winters of the three previous years. This shows an increase of 85 per centum for the epidemic season as compared to the average number of instances for three non-epidemic seasons. The tracings in the chart show in a striking manner how prevailing influenza multiplies the instances of pneumonia. (Chart IV.)

The mortality-rate in influenzal pneumonia was also much higher than during seasons when influenza did not prevail, but I cannot go into details relative to this at present writing.

I have not attempted to differentiate croupous from broncho-pneumonia, since they were found to be unclassified with reference to the particular variety, in the records of the hospital.

PRIMARY CARCINOMA OF THE PANCREAS, WITH MILIARY CARCINOSIS.

By DAVID BEVAN, M.D.

Carcinoma of the pancreas, whether primary or secondary, is usually of the hard or scirrhus variety. Squamous and columnar cell epitheliomata, colloid and encephaloid carcinomata are of extremely rare occurrence, very few such tumors having thus far been reported.

The head of the pancreas is the usual seat of the disease, though in a few instances the cancerous growths have been found restricted to the body or tail. Occasionally the growth will spread from its original site and involve the whole organ.

When primary, the cancerous growth consists in an aggregation of nodules, varying in size from a pea to a large walnut, but when secondary it is simply one large, dense mass.

It occasionally happens that the duct of Wirsung becomes occluded, when retention cysts are developed containing a yellowish-red liquid. When the disease is confined to the head the remainder of the gland may continue normal, though it is more likely to undergo fatty degeneration or to become indurated and atrophied.

Secondary carcinoma, although quite rare, is more frequently met with than primary, and is supposed to occur in about 6 per cent. of all cancer cases. In 11,492 autopsies conducted by Segre, 132 cases of tumor of pancreas were noted, of which number 127 were cancers.

In carcinoma of the pancreas we frequently observe an abundant development of metastatic nodules throughout the abdominal cavity. This would seem to indicate a wide dissemination of the etiological factor, and especially characterizes this form of carcinoma. Of the 127 cases observed by Segre this widespread development of metastatic nodules was absent in but 12.

The carcinomatous growth develops usually after the fortieth year; a notable exception to this rule is the case reported in "Pepper's System of Medicine" of carcinoma in the pancreas of a fœtus.

Men are more frequently affected than women, and, from the reports of cases, the relative proportion would seem to be about 1.5 to 1.

One of the peculiar features of the disease is the fibroid hyperplasia manifest in viscera which show no carcinomatous involvement. The ductus communis choledochus may become occluded, either by being involved in the tumor mass or by pressure. The aorta may undergo fibroid changes and its lumen be greatly diminished, though if the tumor press upon it, dilatation above the point of pressure will ensue.

The progress of the disease is very rapid; the subcutaneous fat quickly disappears; atrophy of the skeletal muscles becomes extreme, and when death takes place—which it does within from one to two years of the onset—the body is excessively emaciated. The muscular tissue is usually of a dark slate color and very friable. A not infrequent association is ascites or anasarca, induced by pressure of the tumor mass upon the abdominal veins. In carcinoma serous transudations are often blood-stained.

The following post-mortem notes were taken at an autopsy, conducted by myself, at the Philadelphia Hospital:

Fanny C——, white; fifty years of age. Clinical diagnosis—carcinoma of stomach. The body is extremely emaciated; rigor mortis is absent, suggillation slight and limited to posterior aspect of trunk; green discoloration of abdominal wall immediately over the iliac fossæ; small bedsore, one inch in diameter, over lower part of sacrum. Subcutaneous fat absent from abdominal wall; recti muscles thin and ribbon-like and of a dark slate color.

The abdominal cavity contains two fluid ounces of a blood-stained serous liquid. A large nodular mass is observed, having its centre in the umbilical region, and, apparently, involving all of the abdominal viscera.

The left pleural sac contains seventeen ounces of blood-stained serous fluid. There are no adhesions. The right pleural sac contains twenty ounces of blood-stained serous fluid. Old and dense adhesions between the middle lobe of lung and chest wall. Pleura opaque and slightly thickened.

The pericardium contains two drachms of clear, serous fluid, and is apparently normal.

The walls of the heart are all relaxed. The right auricle contains one ounce of fluid blood and a small white clot. The right ventricle contains two ounces of fluid blood. The left auricle and left ventricle each contain a small quantity of fluid blood. The muscle of the heart is of a dark slate color and very friable. The valves are all competent and seemingly normal.

The posterior portion of left lung is heavy and dark red in color from hypostatic congestion. The lung is œdematous throughout. The bronchi and bronchioles are intensely hyperæmic and contain considerable quantities of light frothy mucus. The right lung is in about the same condition as left.

The spleen is small; the capsule tense. On the posterior surface approaching

the hilus is a dense white nodule three-fourths of an inch in diameter. Another nodule, of the same character, but slightly smaller, is situated at the upper part of the spleen immediately subjacent to the suspensory ligament. On section, the organ is of a dark chocolate color; the parenchyma soft and the stroma prominent. The two nodules just referred to penetrate into the spleen; and the parenchyma, immediately about them, is of a somewhat firmer consistency than elsewhere.

Both kidneys show those lesions peculiar to interstitial nephritis, but are otherwise unaffected.

The supra-renals are grayish-red in color, and show several small hemorrhagic areas. They are quite firm in texture. The left shows a small, dense, white nodule, about the size of a pea, upon its upper surface.

The upper posterior surface of the bladder is thickly studded with small, dense, white, metastatic nodules, which seem to have their seat in the tissue immediately subjacent to the reflected fold of the peritoneum. The mucous membrane is slightly thickened and congested, and is covered by a thin layer of glairy mucus.

The uterus and its appendages are all seemingly normal, except the right ovary. The latter consists of two distinct parts—the external, a cyst, one inch in diameter; the substance within the capsule is gelatinous, amber in color and perfectly transparent; the internal portion a dense, white nodule, three-fourths of an inch in diameter.

The duodenum is embedded in the large tumor mass before referred to. Its mucous membrane is congested and coated with mucus; its lumen is normal. The serous coat cannot be differentiated from the surrounding tissue. The pyloric extremity of the stomach is also involved in this large central growth. Near the pyloric extremity several nodules about one-half inch in diameter lie in the tissue just beneath the serous coat. The mucous membrane of the whole stomach is intensely red and covered with a tenacious glairy mucus. None of the nodules project into the stomach. The pyloric orifice is unobstructed and apparently undiminished.

The head of the pancreas consists of an aggregation of dense nodules, between which pass strong bands of fibrous tissue. The diameter of this mass is three and one-half inches. On section, it is opaque; and in color white with a grayish tinge. The duct of Wirsung does not seem to have been occluded. The body and tail are indurated and grayish-yellow in color.

The color of the liver is bright red. Its anterior margin is slightly rounded. On the under surface of the liver are to be seen several small, dense, white nodules. The under surface of the diaphragm is thickly studded with these nodules, which vary in size from a mere point to a pea. Although they extend down into the suspensory ligament as far as its attachment to the liver, they have not developed on the superior surface of the latter.

The coils of small intestine are so firmly matted together that it is impossible to separate them. However, small metastatic nodules are to be seen, developing at short intervals in the wall of the intestine, beneath the serous coat.

The upper border of the transverse colon is wholly involved; and it, with the pancreas, omentum, pylorus and duodenum, constitutes the bulk of the central mass.

The aorta is rigid and atheromatous. It is slightly and uniformly dilated, from

the ascending portion of its arch to a point corresponding to the first lumbar vertebra, where it is pressed upon by the tumor mass.

The diagnosis of primary carcinoma of the pancreas is beset with difficulties. It is quite likely to be mistaken for primary carcinoma of the pylorus. This is not surprising when we consider the frequency with which the stomach is secondarily involved early in the progress of the disease. The widely distributed collateral alterations, in the tissue and functions of neighboring viscera, develop a confusion of symptoms well calculated, in some instances, to lead astray the most acute diagnostician.

The neoplasm developing in the head of the pancreas, almost invariably induces, by pressure, some degree of obstruction in the ductus communis choledochus, which engenders a jaundice more or less severe. Symptoms of indigestion are present almost from the onset, and, as the disease advances, become greatly aggravated. The interference with the processes of nutrition leads to a progressive deterioration of the blood, which, taken in conjunction with the tissue changes in the alimentary appendages, results in the elaboration of greatly impaired secretions. This association of nutritive disturbances often develops an exceedingly capricious appetite. At times there is manifested an insatiable desire for food, and whatever is placed before the sufferer is attacked with ravenous greediness and swallowed without an attempt at mastication. In an autopsy, recently conducted by Dr. Coplin, upwards of a dozen prune stones and divers other indigestible substances were found in the stomach.

The secretory glands are not the only ones which sluggishly perform their perverted functions, but the emunctories are affected as well. The kidneys may gradually fail to excrete; the depurative function of the liver may be in abeyance; the glands of the intestinal mucosa may be inactive. Constipation is invariably associated with the two latter conditions. The functional activity of the brain may be greatly impaired, and the patient finally pass into a stupid, moribund condition, largely due to the retention in the blood of the toxic products of tissue metabolism.

REPORT OF SOME INTERESTING CASES IN MY SERVICE IN THE SURGICAL WARDS DURING 1894.

By JOHN B. DEAVER, M.D.

The following cases comprise a number of interesting surgical affections occurring in my wards:

CHOLOCYSTOTOMY, WITH REMOVAL OF A STONE FROM THE COMMON DUCT.

M. H., aged sixty; female; German. Admitted September 29, 1894, under the care of Dr. S. Solis-Cohen, with history of vomiting and pain over liver.

The patient had had several paroxysms of acute pain in abdomen, reflected to the left shoulder and back, lasting a few moments. There was tenderness on pressure over the liver, which extended two inches below the ribs. The urine contained bile, but no albumin. The patient was exceedingly jaundiced; the stools were of a light-yellow color. The temperature ranged between 101° F. and normal. Diagnosis, biliary calculi.

Transferred to surgical ward for operation, October 30, 1894.

Operation, Wednesday, October 31st, before the class. An incision six inches in length made to the inner side of the right semilunar line extending downward; this was subsequently enlarged to eight inches, owing to the great amount of superficial fat. After opening the peritoneum the gall-bladder was easily found. It was free from adhesions.

Digital examination of the gall-bladder and ducts at this point disclosed the presence of one large and several small stones in the bladder, but failed to discover any obstruction in the ducts. The gall-bladder, after being surrounded by gauze, was opened at the fundus and held by two sutures. About twenty stones and fragments were removed, the largest measuring one-half an inch in diameter. This procedure was accompanied by the escape of an ounce or two of bile, some of which was secured for a bacteriological examination.

The space gained after evacuation of the gall-bladder enabled me to pass my finger through the foramen of Winslow, when the presence of a stone one-half inch in diameter was detected in the common duct, near the junction with the duodenum. After enlarging the parietal incision in the upward direction, I was able to pass my finger partly behind the stone, but sufficient fixation could not be obtained to open the duct. After repeated attempts this was accomplished in the following manner: The intestines were walled off from the field of operation by gauze packing, and held out of the way by a special retractor devised by myself for use in abdominal work. This was passed into the wound, and (with the aid of the illumination from an electric headlight) so placed that the end of the retractor was beneath the duct. The finger was then able to grasp the stone and hold

it in position. With the finger as a guide, a scalpel was carefully passed down and an incision an inch in length was made in the duct in the line of its long axis. By aid of the forceps the stone was broken and removed. The retractors were withdrawn, a drainage-tube passed into the duct and surrounded by gauze which was left *in situ*. The fundus of the gall-bladder was anchored to the edges of the parietal wound and drained in a like manner. The external incision was closed by deep sutures of silkworm-gut and superficial sutures of silk, and an aseptic dressing of gauze and cotton placed over the whole. This was changed in three hours, and was found saturated with bile and slightly blood-stained. The second dressing was changed in six hours, and showed only bile.

Bulky and frequent dressings were required, as they were rapidly saturated with bile. The wound united; the gauze packing was removed at end of first week, leaving a biliary fistula, which discharged freely. The stools were large, formed, and clay-colored; the jaundice disappeared. The patient fell into semi-comatose state on tenth day after operation, from which she never roused. Died on November 20th. No autopsy.

A bacteriological examination of contents of gall-bladder was made by Dr. Albert A. Ghrisky, who reports as follows:

About 2 c.cm. of blood-stained fluid bile was secured at the time of operation and immediately examined.

Microscopic cover-slip preparations of the fresh bile showed numerous blood-corpuscles, granular débris and a few bile-stained cells, single and in clumps.

No bacteria were found in the stained preparations examined microscopically.

Inoculations made in glycerine agar-agar, kept in the thermostat at the body temperature, proved negative.

SUBCUTANEOUS OSTEOTOMY FOR DEFORMITY FOLLOWING COLLES'S FRACTURE.

J. F., aged forty-four, white, bricklayer, was admitted October 13, 1894, for angular deformity following Colles's fracture. On December 12th a subcutaneous osteotomy was performed before the class. The deformity was corrected, the wound closed with catgut sutures, an aseptic dressing applied and the forearm enveloped in a plaster dressing. Recovery was uninterrupted and the result perfect.

RAPID DILATATION AND PERINEAL CYSTOTOMY FOR STRICTURE.

P. K., aged fifty, white, born in Ireland. Admitted September 27, 1894. The patient had suffered from gonorrhœa during the war. On admission he had retention of urine of one week's standing. The patient had to be catheterized five to six times daily. Examination showed a firm and resistant stricture at five and one-half inches from the meatus, for the cure of which gradual dilatation was instituted. This was continued, but the patient developed a cystitis, and on December 5, 1894, rapid dilatation up to No. 30 F., followed by perineal cystotomy, was performed. The patient was in such an anæmic condition that he reacted from operation slowly. Stimulation with strychnia, digitalis and whiskey was necessary. A soft rubber catheter was passed through perineal opening into the bladder which was washed out daily with boric acid solution.

Under this treatment he slowly improved. No rise in temperature occurred. The cystitis rapidly subsided and the perineal wound closed slowly. The stricture was kept dilated up to 32 F. There was rapid gain in strength. Discharged, cured, March 19, 1895.

CHRONIC APPENDICITIS.

CASE I.—S. S., colored, single, aged thirty-six years, was admitted October 15, 1894, into the male medical wards of the hospital, care of Dr. W. E. Hughes. Family history negative. The patient used alcohol moderately and tobaceo freely. He had suffered from pleurisy when a child. Three weeks before admission he complained of pain in abdomen, for which he took the usual household remedies without relief. The pain was always worse after eating, sometimes dull, at other times sharp, shooting and burning in character, "like a hot iron in his stomach." He did not vomit, but suffered more or less during the illness from diarrhœa. On the 12th of November I saw the case with Dr. Hughes, and agreed with him in the diagnosis of chronic appendicitis and advised operation. Accordingly, after the usual preparation, the patient was taken before the class November 14th. The operation was performed, and an adherent and diseased appendix, with a small collection of pus between the layers of the meso-appendix, was found. After operation the patient complained of but little pain. The dressings were changed as required and the wound allowed to heal by granulation.

The nourishment consisted of liquid peptonoids, beef-tea, peptonized milk for first ten days, then milk toast, etc., and the medicines, of citrate of potash, quinine and assafœtida suppositories. Discharged, cured, February 7, 1895.

CASE II.—T. R., Italian, aged thirty-six, was admitted October 8th into the male medical wards. His father died of phthisis; family history otherwise negative. He had the usual diseases of childhood, including scarlet fever, and typhoid fever when a young man. About two months before admission he complained of dyspeptic symptoms, with dull pains about abdomen, especially marked in the region of the right iliac fossa. On inspection of the abdomen a tumor was seen to occupy the right iliac fossa, which was tender on pressure.

The patient was emaciating and gradually losing strength. The temperature ranged from 100° to 102° F. Examination of urine was negative.

On October 17th I saw the case and advised early operation. Accordingly, the patient was transferred to my wards. After the usual preparation he was taken before the class October 24, 1894, and the operation of removal of the appendix was performed. After the operation there was some pain for the first forty-eight hours, which completely disappeared at the end of that time. The temperature after the operation was normal.

The post-operative treatment consisted of assafœtida suppositories and bitter tonics. Discharged, cured, January 6, 1895.

URETHRAL STRICTURES; PERINEAL SECTION WITHOUT A GUIDE.

G. N., white, single; occupation, laborer; aged forty-four years; was admitted December 14, 1894.

Family history negative. At the age of twenty-two, while landing a vessel, he fell astride the edge of a coal bunk, striking on his perineum and rupturing his urethra in its membranous portion. For this he was treated at once in Norfolk, Va., by the operation of perineal incision with a guide. A metal catheter was

left in the urethra for ten days. The perineal wound healed rapidly. Attempts to pass instruments after the removal of the catheter were unsuccessful, and a second operation performed six months later. As in the primary operation, an instrument was left in the urethra. The perineal wound healed promptly. The condition of the urethra was not very much improved. At the time of his discharge a No. 8 English instrument was passed with great difficulty. Five years later there was obstruction to urination, for which he was treated in Chicago, when only a filiform bougie could be passed. From that time until December 12, 1894, an interval of sixteen years, he has had frequent attacks of difficult urination, requiring him to apply for relief at dispensaries. There was constant dribbling of urine, which soiled his clothes and was a great source of discomfort to him.

Present Condition.—Urinates every half hour; coustant dribbling of urine; corkscrew stream. Examination of the urethra reveals impermeable stricture of membranous portion.

December 12, 1894.—Perineal section without a guide by dissection was performed. A perineal tube was inserted into the bladder and a soft catheter was passed through the anterior urethra and out of the perineal wound. The bladder tube was removed on sixth day and the urethral tube on the same day. On January 14, 1895, a No. 11 F. was passed. This was followed by a violent epididymitis of right side.

February 7, 1895.—Swelling of testicle diminishing; instruments again passed.

February 11.—Nos. 12 and 13 passed.

February 14.—Nos. 15 and 18 passed.

February 19.—Nos. 21 and 24 passed.

February 26.—Nos. 24 and 26 passed.

March 1.—Perineal wound almost entirely closed, a drop or two of urine passing from it only on rare occasions. The opening of the fistula could not be fouud. Still some swelling of the testicle; No. 26 F. passed with ease. March 20, 1895, discharged. Fistula closed entirely; No. 26 F. passed easily.

EXCISION OF KNEE.

R. F., Italian, aged thirty, admitted July 13, 1894, complaining of pain in right knee.

The joint was swollen, red, and painful on pressure and on motion. The temperature on admission was 101° F. It fluctuated to normal.

On July 16th the knee was opened by a colleague and drainage-tubes introduced through the joint. The knee was dressed daily; free discharge of pus.

The patient came under my charge October 15, 1894, when there was evidence of necrosis of the condyles of the femur. He was prepared for operation October 17, 1894, and resection of the knee-joint was performed.

The inner condyle was the seat of necrosis, and the head of the tibia, of a superficial abscess. The synovial membrane was thickened. The joint surfaces were excised. The wound was closed with silkworm-gut sutures and dressed aseptically, and enveloped in a plaster cast.

The temperature after the operation was normal, and rose for three evenings to 100° F., and then fell to normal, where it remained.

At the end of the sixth week, the plaster cast was removed, and the patient was discharged, cured, January 2, 1895.

URETHRAL STRICTURE; FORCIBLE DIVULSION FOLLOWED BY URINARY EXTRAVASTATION; PERINEAL SECTION WITH A GUIDE; DEATH FROM CROUPOUS PNEUMONIA.

T. G., white, aged forty-eight; occupation, laborer; family history, negative. Nothing known about patient except that he had a stricture for some time.

December 13, 1894, the stricture was located at the bulbo-membranous junction. Under ether, divulsion was performed, and sounds were passed up to No. 25 French. During the next three days he had several chills. The morning after operation the catheter, which had been tied in, came out. This accident was followed by œdema and swelling of the penis and skin of the abdomen and scrotum. This state of affairs continued until the 20th, operation having been declined up to that date.

On that date perineal section with a guide was performed, and free incisions were made into the scrotum, penis and abnominal walls, which were much infiltrated with pus. Drainage-tubes were inserted in several directions from above the pubis to the lower part of the scrotum on each side. Perineal drainage of the bladder was used.

After the operation the temperature rose to 103°, with intermissions of two to three degrees for the following four days. On December 24th a croupous pneumonia developed in the left lung, from which the patient died on December 27th.

INTERESTING CASES OCCURRING IN THE SURGICAL DEPARTMENT OF THE PHILADELPHIA HOSPITAL,

EMBRACING A CASE OF SUCCESSFUL LIGATION OF THE THIRD PORTION OF THE SUBCLAVIAN ARTERY FOR THE CURE OF AXILLARY ANEURISM; TWO CASES OF APPENDICITIS; ONE CASE OF TUBERCULAR PERITONITIS; TWO OPERATIONS FOR THE RADICAL CURE OF HERNIA; A CASE OF SUPRA-PUBIC CYSTOTOMY, MADE NECESSARY BY RETENTION OF URINE CAUSED BY A HYPERTROPHIED PROSTATE; AND AN OPERATION FOR THE REMOVAL OF A BRASS RING AROUND THE ROOT OF THE PENIS.

BY ORVILLE HORWITZ, M.D.

As my contribution to the HOSPITAL REPORTS for the current year, I have selected a few cases from the Surgical Department possessing interesting features.

SUCCESSFUL LIGATION OF THE THIRD PORTION OF THE SUBCLAVIAN.

Probably the most common causes for the formation of aneurism of the axillary artery are over exertion and traumatism. The close proximity of the tumor to the heart, and the loose connective tissue surrounding it, predispose it to very rapid growth, with all the attending dangers of rupture; consequently, early and energetic treatment is indicated.

It is essential that an operation should be performed as soon as possible after the diagnosis has been established. Barwell has shown that when the aneurism is purely axillary, three out of four cases treated by ligation of the third portion of the subclavian artery recover; but if allowed to remain, the aneurism is apt to change to the subclavio-axillary variety, in which case the mortality from operation is greatly increased. Ninety cases of the latter description, in which the ligature was employed, are tabulated in the "International Encyclopædia of Surgery;" out of this number twenty-seven died, making a mortality of 35.5 per cent.

The methods of treating axillary aneurism are diet and rest, manipulation, proximal pressure, distal and direct pressure, galvano-puncture, injection of the perchloride of iron, and, finally, ligation of the third portion of the subclavian artery.

The first two methods are unsatisfactory, and we fail to find any cases reported where they have been employed successfully. Proximal pressure more frequently fails than succeeds; it is a very difficult form of treatment to properly carry out. It might be tried when the aneurism is seen early, and is very small. Distal and direct pressure have likewise failed. Galvano-puncture might be employed in those conditions which render ligation impossible. The use of perchloride of iron by injection should be looked upon as a very dangerous expedient, not to be recommended. This leaves ligation as the most certain and the best method of treatment that can be employed.

The results of 16 cases of aneurism of the axillary artery, treated in six of the London hospitals, are as follows: 5 cases by pressure, 3 failures, 1 death, 1 recovery; 3 amputations, 3 deaths; 1 case by Valsalva's method with aconite, no benefit; 3 cases by expectant measures, 3 deaths; 4 ligations, 3 recoveries, 1 death.

After briefly stating these preliminary statistics, I submit the following case:

Edward Deaver, a laborer, forty-eight years of age, was admitted to the Philadelphia Hospital on the 1st of April last, complaining of pain and loss of power in the right arm.

The patient was not addicted to alcohol, neither had he been the victim of syphilis or rheumatism. His family history was negative. His heart, lungs, and kidneys were normal; there was a slight arcus senilis.

He stated that two months previous to his admission to the institution he began to suffer from pain in the right arm, which was neuralgic in character and most marked in the neighborhood of the elbow-joint. Occasionally the pain would dart down into the hand and fingers. The pain gradually increased in severity, and was aggravated by using the arm or allowing it to hang by the side of the body. At times he was obliged to raise the arm above the head in order to obtain relief from this troublesome symptom. Shortly after the onset of the pain a tumor appeared in the axilla, which rapidly increased in size.

On examination, a fullness was discovered beneath the clavicle; the axillary space was occupied by a swelling about the size of an orange, which, on palpation, was found to be the seat of an expansile pulsation. It was pseudo-fluctuating and compressible. The radial pulse on the right side was markedly diminished in volume in comparison with that of the left. On auscultation over the tumor a loud bruit was clearly discernible. There was considerable œdema of the hand and wrist. It was apparent that an aneurism existed, and ligation of the third portion

of the subclavian artery was decided upon. On the night before the operation the patient was prepared in the usual manner.

The Operation.—Having placed the patient on his back, his head drawn to the left side of the body, and his shoulders raised, his arm was pulled down and the fore-arm pushed well under the back, after the manner suggested by Treves, who claims that placing the patient in this position pulls down the clavicle and increases the size of the subclavian triangle.

In order to avoid wounding the external jugular vein, the integuments of the neck were drawn well down below the clavicle ; an incision, three inches in length, was made along the posterior border of the clavicle, extending one inch from the sternal end of the bone to the border of the trapezius muscle. The direction of the cut was so made that the incision ran just above the upper border of the clavicle, and the knife was drawn along the bone, and the instrument made to penetrate to only one-half the depth of the clavicle. By this means the layer of fascia which runs from the sterno-mastoid to the trapezius muscle was divided, after which, following the suggestion of Barwell, the rest of the dissection was completed by means of the dry dissector.

The transversalis colli vein was found distended and much in the way, but was held to one side by means of a blunt hook. The scalenus anticus muscle having been laid bare, the tubercle of the first rib was easily located as a guide to the artery, which was discovered in company with the lower cord of the brachial plexus, which was lying upon and attached to the vessel. The nerves were separated from the artery and drawn to one side, bringing the sheath of the vessel into view. The artery was then freed from the connective tissue surrounding it by means of the index finger. The sheath of the artery was carefully torn through by the aid of a grooved director, when the wound became immediately filled with blood ; the index finger was carried to the bleeding spot, and the part thoroughly sponged, when it was discovered that a small vessel in the sheath of the artery had been wounded. The bleeding was easily controlled by means of a pair of hæmostatic forceps. It was with some difficulty that the aneurism needle could be made to pass around the artery owing to the fact that the vessel was lying deeply under the clavicle, and that the sheath was adherent to the wall of the vessel. The artery was ligated with a stout silk ligature. The wound was closed by means of interrupted silkworm-gut sutures, no drainage being employed.

Immediately after the ligation the radial artery ceased to beat, the arm assumed a peculiar mottled appearance, and became cold to the touch. The wound was dressed antiseptically, the arm wrapped in cotton batting, lightly bandaged, and elevated on a pillow.

Within twenty-four hours the radial pulse began feebly to beat. The sutures were removed by the eighth day, union having taken place by first intention. Shortly afterwards the patient was discharged cured.

In reporting this case the writer wishes to call attention to certain points of interest in connection with it.

By following the suggestion of Barwell—that is, after dividing the fascia which passes from the sterno-mastoid to the trapezius muscle not again to employ the knife during the operation—the capillaries and small vessels lying in the connective tissue over the

vessel were torn instead of being cut, and the hemorrhage, which otherwise would have obscured the field of vision and delayed the operation, was prevented and the isolation of the artery was rendered much easier.

The best deep anatomical guide to the subclavian artery is the lower cord of the brachial plexus, which not only lies directly over but is attached to the sheath of the vessel. When the nerve was first uncovered it was affected by a transmitted pulsation from the artery, and in appearance resembled it. It was, however, distinguished by its resistance, and by the fact that when it was compressed between the index finger and thumb no dimunition in the volume of the radial pulse resulted.

It will be observed that it was stated that the aneurism needle was passed with difficulty. Whenever this is the case, it is probably caused by the sheath of the artery being intimately adherent to the outer coat of the vessel. When demonstrator of surgery in Jefferson Medical College, I frequently observed that students in the surgical laboratory experienced great difficulty in passing the aneurism needle after the artery had been exposed. Whilst assisting a surgeon in this city some years ago in the ligation of the common carotid artery, the same trouble in passing a needle was experienced; suddenly, on using a little extra force, the instrument slipped under the artery and wounded the jugular vein, so that it became necessary to ligate both vessels.

The wounding of the small vessel while opening the sheath of the artery was not only unexpected, but, to a certain extent, alarming. We were under the impression that the location of a vessel in that situation must be most unusual. While witnessing a similar ligation by Professor Keen, preparatory to the amputation of the upper extremity, including the clavicle and scapula, on tearing through the sheath of the subclavian artery, I noticed that the same accident occurred.

These two cases lead us to infer that this condition may not be as unusual as is generally supposed. It is mentioned here so that any future operator meeting with a similar experience may not be unduly anxious. The sudden appearance of blood after opening the sheath of a large artery like the subclavian, coming from a deep, well-shaped wound, the bottom of which not being over half an inch long, might be the source of unnecessary anxiety unless the origin of the sudden hemorrhage is thoroughly understood.

TWO CASES OF APPENDICITIS.

During the last part of the month of April I was sent for to meet Dr. Frederick Packard in consultation as to the advisability of an immediate operation in a case of appendicitis. It was midnight when I reached the hospital, and found the following conditions:

CASE I.—The patient was a laborer by occupation, aged thirty-two. He had been in the hospital twenty-four hours; he had been sick five days previous to his admission; he was suffering with nausea and vomiting, attended with severe pain in the neighborhood of the epigastrium, most marked in the right iliac fossa. The parts were distended, and so tender that a thorough examination could not be made by palpation. The seat of greatest tenderness was over McBurney's point. His bowels had been moved several times since admission. The tongue was dry and coated; temperature, 103° F.; skin moist. He stated that this was the second attack of the kind from which he had suffered within the previous three months.

Physical examination convinced me that the individual was laboring under an acute attack of appendicitis, attended with suppuration. It was evident that an operation should not be delayed.

On opening the peritoneum a large quantity of fœtid pus escaped. The cavity was irrigated with hot distilled water and carefully examined, when a circumscribed abscess presented itself, but the appendix was not seen. Deeming it inexpedient to make an extended search, lest adhesions should be broken up, and the general peritoneal sac become infected, the cavity was simply packed with iodoform gauze, and dressed in the usual manner. The patient made a rapid recovery.

CASE II.—The second case was admitted into the hospital suffering with acute appendicitis, which had lasted for the previous forty-eight hours. When first seen by me, the patient complained of great pain in the right iliac fossa; the abdomen was distended, temperature 102° F., tongue dry and furred. His bowels not having been moved since the beginning of his attack, Epsom salts, in doses of one drachm every two hours, with occasional small doses of opium to alleviate the pain, were ordered. Under this treatment his bowels were moved several times during the day, but the general condition was worse, and an immediate operation became necessary.

On reaching the appendix it was found to be enlarged, adherent, and containing pus. It was at once removed, the stump buried in the cæcum in the usual manner, and the wound closed. The patient recovered rapidly.

As a rule the diagnosis in a case of acute appendicitis is not difficult; in chronic cases, the position and condition of the appendix may frequently be obtained by palpation.

Each case must be a law unto itself as regards the most favorable time for operating; it may be conceded, however, that it is far safer to operate early than to lose time by dangerous procrastination. In acute cases, if after the administration of purgatives the symptoms do not yield, or the local tenderness is increased, an operation should be performed without delay.

Much difference of opinion exists as to the length of the incision in cases of suppurative appendicitis. A safe rule is to make a small incision, but sufficiently large to allow the pus to escape, irrigate the cavity, explore it by means of the index finger, and then obtain a view of its walls by means of an electric headlight.

Operations on circumscribed abdominal abscesses are among the simplest in surgery. It is remarkable what large cavities may be drained by means of small incisions, and the patients make a rapid recovery.

There are two points to be borne in mind, first to make the incision large enough to permit free exit to the pus, and to allow of the exploration of the cavity, and, secondly, to make the smallest incision that will insure free drainage. On the carrying out of these rules depend the rapid and perfect recovery of the patient.

It seems to be a well-established fact that in operating upon these abscesses it is safest not to run the risk of breaking up adhesions by searching for the appendix, and thereby run the risk of entailing general peritonitis. In most of the cases that I have witnessed, when the operator has persisted in hunting for and removing the appendix, the result has been peritonitis and death.

TUBERCULAR PERITONITIS.

The third case is of interest from the fact that its history, obtained from the patient, and the general symptoms seemed to indicate that suppurative appendicitis was present, but on examination it proved to be one of tubercular peritonitis. The patient was a foreigner, and the statement of his case was given with great difficulty.

The patient had been in the hospital two days when I was summoned. He was a laboring man, thirty-nine years of age ; had been sick for ten days previous to his admission. He was suffering from colicky pains in the region of the epigastrium, attended with nausea and diarrhœa. There was general abdominal tenderness, especially marked in the right iliac fossa. Respiration was increased in frequency ; tongue heavily coated; temperature, 102° F. The lungs, heart and kidneys appeared to be normal. Under treatment he had become progressively worse. The abdomen was much distended, very tender to the touch, and the iliac fossa on the right side contained a doughy mass, which was very sensitive. The pain was most marked over McBurney's point.

An operation was decided upon, and on opening the peritoneum a large amount of white serous liquid, intermixed with flakes of lymph, escaped. The appendix was normal, and the ilium was adherent and bound down to the colon, which explained the presence of the doughy mass that had been observed before the

operation. The intestines were studded with tubercles. The bowels were not distended with gas.

After washing out the abdominal cavity with hot water, it was dusted with sterilized iodoform, and a glass drainage-tube was inserted.

The patient survived the operation two days, and died, apparently from exhaustion; during this time he suffered from uncontrollable diarrhœa, accompanied by attacks of vomiting.

It is probable that the patient had been ill for a much longer period than he stated when admitted into the hospital, though he possibly may not have understood the question bearing upon this point, or he may have dated his illness from the time that his symptoms had become serious.

An interesting condition, in connection with this case, was the great distention of the abdomen without the presence of gas in the intestines; it was out of all proportion to the amount of liquid which escaped through the incision. I have frequently seen this state exist in cases of peritonitis upon which I have operated, and have expected to find the bowels much distended with gas, yet, on cutting through the peritoneum, they have proved to be in a normal condition. When the abdominal distention is associated with inflammation of the parietal peritoneum and the bowels are distended, the percussion-note is much flatter than when the intestines are distended with gas.

SUPRA-PUBIC CYSTOTOMY.

In the fourth case supra-pubic cystotomy became necessary in order to relieve an overdistended bladder, caused by an hypertrophied prostate.

The patient was a tailor, sixty-nine years old. When first seen he had not passed water for twenty-four hours. A rectal examination discovered an enormously large prostate. The bladder was distended very nearly to the umbilicus; he was suffering greatly.

Before he entered the institution repeated efforts had been unsuccessfully made to draw off the water by means of the catheter. A few drops of blood had oozed from the meatus. The passing of an instrument of any kind was impossible.

The bladder was aspirated and the patient placed in a hot bath; a full dose of quinine and a quarter of a grain of morphia were directed to be given. He was then wrapped in blankets, and allowed to perspire freely.

When next seen he was still unable to pass water, and the bladder was much distended. It was still impossible to pass the catheter.

He was prepared for operation, and brought into the clinic-room. The bladder was distended, which made the procedure very simple; the rectal bag, usually employed, was unnecessary. The usual incision was made, and the bladder exposed, when, on laying my finger on the viscus in order to see if any of the pre-

vescical veins were in the way before catching the walls of the bladder with a tenaculum it passed through the wall, directly into the cavity of the bladder, the urine escaping along the sides of the finger. The prostate gland was found to be a solid fibrous ring. The condition of the patient, together with the increased danger from prostatectomy in cases of ring prostate, decided me to simply establish supra-pubic drainage, which was done in the usual manner.

The patient recovered with a fistulous opening above the pubes, which is kept patulous by inserting through the orifice, once a day, a soft rubber catheter. Most of the urine passes through the fistula, there being no leak unless the bladder is allowed to become overdistended.

RADICAL CURE OF HERNIA.

The histories of two cases of hernia in which the radical cure was attempted are here presented, for it is only by reporting the results of operations of this kind that a true estimate of the value of this method of treatment can be fairly determined.

CASE I.—The patient was brought to the hospital, suffering from strangulated indirect inguinal hernia of the right side. He had labored under the form of reducible hernia for six years, for which he had always worn a truss. The night before his admission, on attempting to lift a heavy stone, the truss slipped and the gut descended into the scrotum. At first he made no effort at reduction, but beginning to feel pain he tried to replace the parts, but failed. He then consulted a physician, whose efforts at reduction by taxis were ineffectual. He was directed to return to his home and apply cloths which had been steeped in cold water; a few hours later the physician made another unsuccessful effort at reduction, when he was advised to go to the hospital.

On examination, the parts were found to be tense, hard, and painful on slight pressure. There was nausea, but no vomiting. The resident surgeon had made several unavailing attempts at reduction by taxis. A final effort, made under ether, was unsuccessful, and the patient was prepared for operation at once.

The gut was found in good condition; a piece of omentum was ligated and removed. The intestines were returned to the cavity, and a radical cure attempted by what is known as the Bassini operation. Silk was employed for the deep sutures in lieu of kangaroo tendon, which was not procurable at the time. Two months later these sutures became troublesome and were removed. The patient made a good recovery, and when seen after the lapse of a year, no return of the hernia had taken place, though the use of a truss was still necessary.

CASE II was a man, fifty years old, who was affected with scrotal hernia of the left side, the ring being very large. Various forms of truss had been employed in futile attempts to retain the parts in place.

The Bassini operation was performed, with kangaroo tendon for the deep sutures. The patient was directed to continue the use of the truss. At the end of six months the hernia again descended, but the ring was by no means as large as before the operation, and the intestines were readily retained in their place by a truss.

In view of the reports of many successful operations by the method pursued by Professor Halstead, I shall, when circumstances permit, resort to the process recommended by that surgeon, *i. e.*, use silver wire for the superficial sutures.

The operation for the radical cure of hernia should not be attempted, save when strangulation occurs; when the operation for the relief of constriction has to be performed; when the hernia is irreducible and gives rise to serious inconvenience, and when it cannct be retained in place by means of a truss.

The operation is not free from risk; a large percentage of cases usually relapse within a year. The mortality is increased when the hernia is very large, in the aged, and in individuals suffering from obesity, heart disease or chronic bronchitis.

STRANGULATION OF THE PENIS BY A BRASS RING.

The next case is of interest, because it is seldom seen by a medical man.

During the month of September last, whilst making a professional visit to the wards of the hospital, I was told that a patient had just been brought in who was suffering from the effect of having slipped a brass ring over his penis.

As it was clinic day, I had the patient brought before the class. He was a young man, aged about twenty-five. He stated that when about to leave his mistress that morning, she had given him a brass ring, and induced him to pass it over the penis to the peno-scrotal junction. To accomplish this feat it had been necessary to lubricate the parts with vaseline. Shortly after going to work he found that the ring was becoming very tight, giving rise to a great deal of discomfort; he examined the part, and was alarmed to find the organ discolored and enormously swollen. The pain by this time had become very distressing, and it was impossible to remove the ring.

I found the penis, owing to œdema, to be double the normal size; it was much discolored, and cold to the touch. The ring had made a deep indentation at the root of the organ. Multiple punctures were made in the skin by means of a tenotome, so as to allow the serum to escape, in the hope that the size of the organ would diminish and the ring could be removed. The effort was unsuccessful, and the patient was etherized preparatory to making an attempt to cut through the ring by means of a file. In consequence of the enormous swelling this could not be accomplished. The ring was finally divided with a pair of pliers; the work was gradually performed by cutting slowly through the band. Then a few incisions were made in different portions of the organ so as to relieve the tension by draining off the serum, trusting in this manner to prevent sloughing.

To obviate retention of urine, a sterilized, soft rubber catheter was passed into the bladder and tied in place. In spite of every precaution extensive sloughing followed. The patient ultimately recovered, the penis being considerably scarred.

In a similar case that came under my care, the cutting-pliers were employed to remove the ring. The catheter was not passed at the time of the operation, and retention of urine, as a consequence, supervened. It was with great difficulty that a very small catheter could be inserted after swelling about the constricted portion of the urethra had taken place. Hence, when the second case presented itself, I was careful to insert a catheter immediately after removing the constricting body, and had every reason to be satisfied with the result.

ATROPHIC SCIRRHUS OF THE MAMMARY GLAND OF OVER FORTY-SEVEN YEARS' DURATION.

By JAMES M. BARTON, M.D.,

AND

D. E. ESTERLY, M.D., Resident Physician.

Catharine H., sixty-eight years of age, was admitted to the Philadelphia Hospital in September, 1894. She is married and has been a housekeeper by occupation. She was born in Ireland, but has resided in Philadelphia for the last thirty-five years. Both her father and her mother died at an advanced age. One sister died after giving birth to a child and another died of unknown cause, at the age of twenty-five. One brother died after a short illness during the War of the Rebellion. Two sisters, older than the patient, are still living and enjoying good health. No history of hereditary disease of any kind can be obtained.

At sixteen years of age, when her menstruation first appeared, the patient was confined to bed for three years; since then her periods have been regular and attended by neither pain nor distress. With the above exception, the patient has during her entire life enjoyed the most perfect health. Her appearance fully corroborates this statement. She looks like a woman of but fifty years of age.

She was married at thirty, but was never pregnant. The menopause occurred at thirty-five.

When she was about twenty years of age, she noticed a small induration, about the size of an ordinary pearl button, under the skin of the right breast. This gradually enlarged until it had attained the dimensions of a hen's egg. It was painless, except during a short attack of inflammation, which occurred soon after its first appearance.

This attack of inflammation was not preceded by any injury that she is aware of. The growth remained stationary for about ten years, when it ulcerated; this ulceration persisted until its removal, in September, 1894. There were many attempts at healing, but before it would completely cicatrize it would open again.

At the time of operation (September, 1894), it was rather larger than a hen's egg and occupied the lower and outer portion of an otherwise wasted right breast; it was very hard and not adherent to the underlying muscle. The ulcerated portion was an inch and a half in diameter; its edges were corrugated and inverted. To the inner side of the growth the skin and underlying tissues were scarred and contracted, looking as though a portion of the growth had once occupied this situation. (See Fig. 1.) There was no involvement of the lymphatic glands, either in the axilla or elsewhere.

The breast was removed by Dr. Barton at the hospital clinic on the 26th of September, 1894. The patient made an uninterrupted recovery. On examination, January 26, 1895, the cicatrix was found perfectly healthy, and there was no evidence, either externally or internally, of any recurrence of the disease.

REPORT OF MACROSCOPIC AND MICROSCOPIC EXAMINATION MADE BY DR. D. RIESMAN.

Macroscopic.—The mass removed at the operation is oval in outline—9 cm. by 5½ cm. wide and 2 cm. thick. It consists of skin and subcutaneous tissue, the latter being infiltrated with fat. About one-half of the cutaneous surface is occupied by a crater-like ulcer, circular in outline. The edges are indurated, irregular, and, in places, inverted. The floor is smooth, except at the upper angle, where there is a small papillomatous mass. A few bands of fibrous tissue traverse the bottom of the ulcer. The nipple is invisible, but at a point 2½ cm. above the edge of the ulcer there is a linear invagination of the skin, 1 cm. long and 11 mm. deep, on superficial examination appearing as the remains of the nipple. It is more likely, however, that the nipple became involved in the growth and was destroyed by it.

Microscopic.—Pieces of the tumor were hardened in alcohol, imbedded in celloidin, and the cut sections stained with hæmatoxylin, hæmatoxylin and eosine, lithium carmine, and picro-carmine.

On examining a picro-carmine section with a low power of the microscope, we find on the surface a layer of squamous epithelium, quite normal in appearance and corresponding to the skin of the mamma, being somewhat peculiar only in the presence of several exceedingly long processes which dip far down into the deeper tissues; but there is no cancerous development in the squamous epithelium. A sebaceous gland and some hair fallicles are also seen. Beneath this surface layer, and separated from it by a dense accumulation of round and epitheloid cells, is a tissue plainly carcinomatous in nature, arranged in the form of large lobules made up of smaller acini. Connective tissue quite rich in round cells surrounds the lobules and sends in delicate partitions. The cells filling these "nests" are distinctly epithelial, and in their general appearance suggest an origin from the acinous portion of the mammary gland.

In the parts of the tumor nearer the center of the ulcer, the connective tissue is more prominent and forms thick fibrous bands, radiating in a peculiar manner through the cancerous tissue and isolating the latter in nests of an unusual shape. We find the tumor tissue running in long winding cylinders between bands of fibrous tissue, the epithelial cells nearest the stroma being columnar in shape. The inner layers consist of the usual polymorphous cells. In some parts of the section the epithelial cells are arranged in a generally circular manner, several layers thick, around a central mass of fibrous tissue, instead of the connective tissue around nests of cancer cells. How this peculiar appearance was produced, I am unable to say. If we trace the connective tissue from its first beginning in the nests just beneath the epiderm down into the part presenting the anomalous arrangement, we can clearly discern, I think, the fibrous tissue has grown *into* the cancer nests. This, of course, does not fully elucidate the cause of the strange relation of cells and stroma.

FIG. 1.

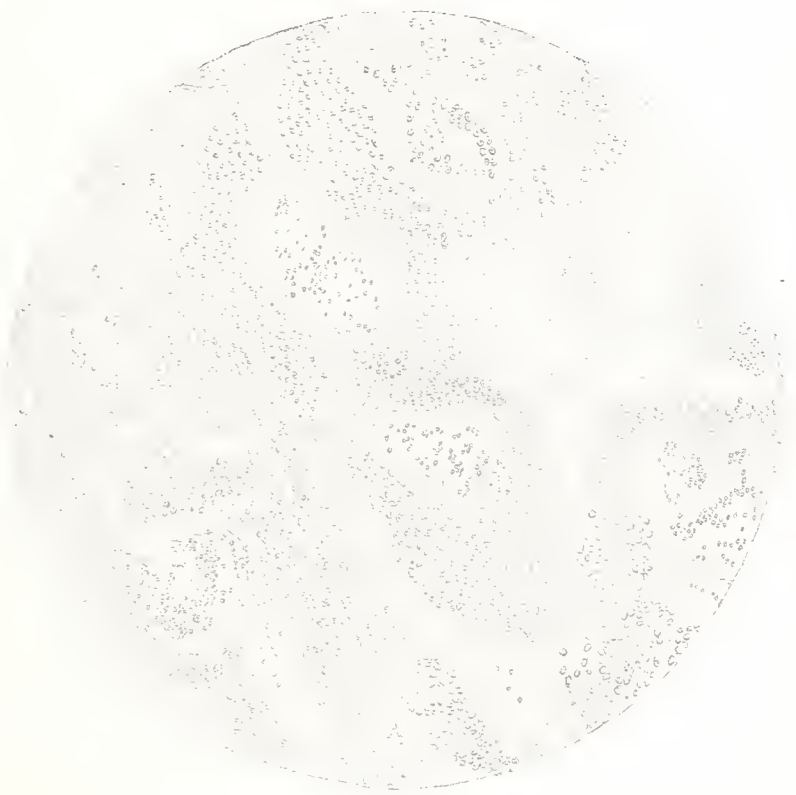
Atrophic scirrhus of the mammary gland.

In the larger nests, the center is, as a rule, degenerated into a granular amorphous substance, but toward the periphery, the unstained outlines of the original cells are still visible. The degeneration is probably fatty.

Some of the nests are entirely devoid of cells; in others, the degeneration is so complete that only a granular débris, staining faintly with picric acid, is visible. In several places the cancer cells are deposited circularly around thickened blood-vessels, causing an appearance analogous to that of the peculiar sarcoma known as *angio-sarcoma tubulare*. In some of the thicker bands of fibrous tissue, ducts are seen on cross-section, their lining being a low columnar epithelium. (Fig. 2.)

The subcutaneous fat is not invaded, at least as far as it is shown in the sections. In a few of the sections, a large, hemorrhagic focus is noticeable; in others, we find extensive deposits of hæmosiderin, the remains of old extravasations.

FIG. 2.



Summing up the features of this tumor, which is peculiar clinically as well as histologically, we would term it a *carcinoma of the breast*, which, in part, is a *carcinoma simplex*, or glandular cancer, in part an *adeno-carcinoma*.

A COINCIDENT INTRA-UTERINE AND EXTRA-UTERINE PREGNANCY.

By BARTON COOKE HIRST, M.D.

During my term of service in the Philadelphia Hospital, in 1894, I removed a tubal-gestation-sac by abdominal section from a young woman with a peculiar history.

Just four weeks before I saw her she had induced abortion on herself, at the fourth month of gestation, by passing into the womb a rubber catheter and its steel stylet that she had bought at a drug store. Profuse hemorrhage followed, and a few hours later the fœtus was discharged. She saw it plainly, distinguished its limbs, trunk, head and face, and from her unprompted description of its development and length it corresponded with the date that she believed pregnancy had reached. She threw the fœtus and its placenta down a water-closet. Shortly after the delivery the woman was seized with abdominal pains that increased in severity until she was forced to go to bed.

When I first saw her I found the temperature 103°, the pulse rapid and feeble, the abdomen tympanitic and exquisitely sensitive. On vaginal examination, large, tender masses were felt behind and to the right side of the uterus. I concluded, naturally enough, that I had to deal with a case of a common kind in this hospital—septic infection of pelvic tissue from criminally induced abortion.

The abdomen was opened the following day, when, to my surprise, I found the conditions characteristic of tubal pregnancy and no sign whatever of septic inflammation. Old clots and a quantity of black blood welled out as soon as the peritoneum was incised, and, on removing the right tube, a gestation-sac of from six to eight weeks' development was found in it, without an embryo, but with the chorial villi so well developed and so evident that there was no mistaking the character of the mass within the tube. This was an extra-uterine preg-

nancy that never could have been diagnosticated or even suspected before operation. The intra-uterine pregnancy that must have coexisted with it and the induced abortion completely masked its symptoms. The patient made a perfect recovery.

A coincident intra-uterine and extra-uterine pregnancy is rare. Gutzwiller,[1] in a thorough search through medical literature up to 1893, was able to find only eighteen cases. Mine is the nineteenth case, and I am unable to find any more. The first case was reported in 1820, so there were nineteen in seventy-five years, a rarity scarcely equaled by even the most infrequent occurrences in obstetric practice.

[1] Arch. f. Gyn., Bd. 43, H. 2, p. 223.

OVARIAN ABSCESS FOLLOWING LABOR; HYSTERECTOMY; RECOVERY; WITH REMARKS UPON THE USE OF GAUZE IN ABDOMINAL SURGERY.

By W. EASTERLY ASHTON, M.D.

The patient was delivered of a healthy child on the first day of March, 1894. The labor was normal, and she did well until the sixth day, when fever and symptoms of post-partum sepsis developed.

I saw the case for the first time on the tenth day after labor, and found the patient in a condition of profound prostration, with the temperature 104° F. and the pulse very rapid and feeble. The abdomen was flat, and no pain was felt upon pressure over the pelvic organs. An examination, per vaginam, revealed a large and flabby uterus which was freely movable; the uterine appendages were apparently normal.

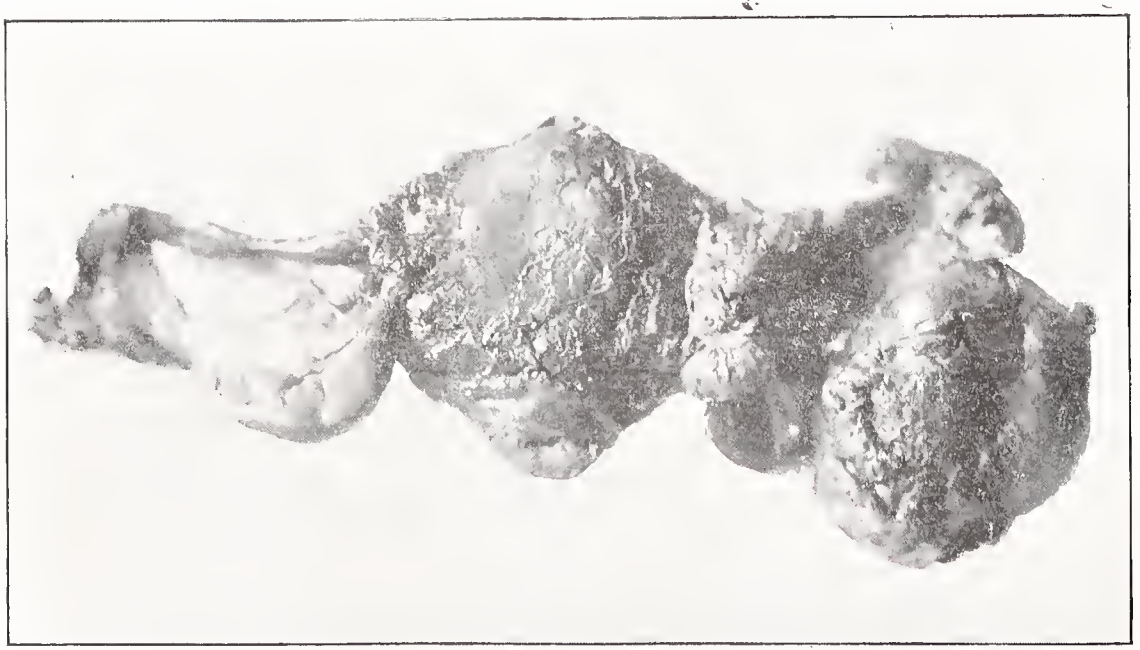
A diagnosis of septic endometritis was made, and the uterine cavity curetted and packed with gauze. Although prompt and decided improvement in the condition of the patient followed this operation, her convalescence was slow and far from satisfactory. Her strength did not improve rapidly; there was a daily rise in the temperature, and she began to complain of pain in the right inguinal region.

I saw the patient for the second time about the 1st of June, 1894. The uterus was found to be fixed in the pelvis and pushed toward the left, while on the right side was felt a semi-fluctuating mass the size of a lemon. Abdominal section was decided upon and performed a few days later.

The omentum was adherent to the bladder, uterus and lateral walls of the abdomen. It was ligated at its lower third and cut away, the resected portion being then freed from its attachments. Before exposing the pelvic organs, it was found necessary to separate a few coils of ileum which had become adherent. The left side of the pelvis was normal, but in the right side an abscess of the ovary the size of a lemon was found closely adherent to the head of the colon and vermiform appendix. (See Figure.) The uterus was enlarged; its walls were very much softened and filled in several places with small collections of pus. After separating the adhesions and ligating the broad ligaments, the uterus and its appendages were removed. In separating the ovarian abscess from the colon, an opening an inch square was made into the bowel. This accident was unavoidable, as the wall of the colon was completely destroyed. The opening into the bowel was closed with silk sutures, and the appendix, which was enlarged and gangrenous, removed.

Some difficulty was experienced in extirpating the uterus, owing to the thickening of the broad ligament on the right side. The position of pelvic elevation was employed throughout the operation, and the general peritoneal cavity protected with gauze pads. Although the abscess-sac ruptured during its delivery, no irrigation was used, the pelvic cavity and surrounding parts being sponged clean with

FIG. I.



Posterior view—Ovarian abscess on the right side of the uterus.

gauze wrung out of hot sterile water. Owing to several ulcerating surfaces left in the pelvis and on the parietal peritoneum after the separation of the ovarian abscess, gauze packing was used to protect the general abdominal cavity. This packing was removed on the fifth day after the operation. The patient made a good recovery.

REMARKS ON THE USE OF GAUZE IN ABDOMINAL SURGERY.

The use of the gauze packing contributed not a little, in my judgment, toward the recovery of the patient. Had glass drainage been employed, post-operative infection would have probably resulted in the death of the patient, as the tube would not have prevented the abdominal viscera from coming in direct contact with the ulcerated surfaces, which were, of a necessity, left in the right side of the pelvis and on the parietal peritoneum.

I have for sometime now given up entirely the use of gauze for the purposes of drainage for the reason that it does not drain and, therefore, does harm when employed with that object in view. I am well aware that this view is contrary to the opinion of most surgeons, yet I have never seen any material, except water or serum, escape through the abdominal wound when gauze drainage was used. Surgeons who use irrigation in making the toilet of the peritoneum overlook the fact that a large quantity of water remains within the abdominal cavity after the operation is completed and the dressings applied. Under these circumstances, if gauze has been used for drainage, the dressings become rapidly saturated, not with blood, pus or septic débris, but with the retained irrigating fluid stained with blood. The same conditions exist also after operations followed by serous oozing. These facts have led us for a long time to believe in the advantages of gauze as a drain when, in reality, it removes from the abdominal or pelvic cavity only those materials which do little or no harm.

In a recent article by Coe upon this subject (*N. Y. Polyclinic*, September, 1895) he says that "he has derived considerable comfort from a recent paper read by Sänger before the Leipsic Obstetrical Society, in which that acute observer absolutely denies the possibility of draining the uterus with iodoform gauze and asserts that it acts simply as a tampon, opposing instead of favoring, the escape of secretions. In the discussion of this paper, Döderlein and Zweifel agreed entirely with this opinion, the former adding that gauze introduced into the peritoneal cavity after coeliotomy acts simply as a tampon, never as a drain."

Dr. Baldy and myself, in our experimental studies in intestinal surgery upon the lower animals, found that all sutures were buried beneath exudate within five hours after operation, and that the approximated surfaces of the gut, following an intestinal anastomosis, were sufficiently firm after four or five days to hold without the support of the rings. These facts give us the principle upon which to base our views as to the use of gauze within the abdominal cavity. The object to be accomplished, after an operation involving the separation of extensive adhesions within the abdomen or pelvis, is not, for the time being, to drain, but to keep the viscera from coming in contact with denuded or septic surfaces and to arrest the oozing of blood. As both of these objects can be accomplished with gauze packing, the question of drainage, when it exists, may safely be left until the packing is removed, when there remains simply an extra-peritoneal cavity, which may be drained with rubber tubing, if necessary. This practice does not apply to certain cases of incomplete operation or to conditions which may exist in appendicitis, for the reason that in these cases it may be necessary to use rubber or glass drainage, either alone or along with gauze packing, on account of the discharges being so free that infection would occur before the general peritoneal cavity could be shut off by the exudate.

In packing either within the pelvis or abdomen, plain sterile gauze is preferable to the ordinary iodoform gauze, as the latter, on account of the glycerine which it contains, causes a profuse serous discharge. The packing should be removed on the fourth or fifth day after the operation, and the extra-peritoneal cavity which remains may be drained and irrigated as the case demands. In placing the gauze packing after a pelvic operation, it is necessary to have the patient placed in the Trendelenburg posture. After the tampon has been placed in position the patient is gradually lowered so as not to disturb the packing and to allow the intestines to accommodate themselves about the gauze.

THE TREATMENT OF SEVERE PNEUMONIA, ATTENDED WITH VERY HIGH TEMPERATURE, IN YOUNG CHILDREN.

By EDWARD P. DAVIS, A.M, M.D.

Although in very young children pneumonia is often a gradual process attended by moderate elevation of temperature for some time, cases are occasionally seen where sudden and excessive fever with intense congestion threaten a fatal issue. The case under consideration illustrates this condition.

The patient, a girl, aged about four years, was admitted to the Philadelphia Hospital during the winter of 1894–95 from a children's asylum. She was reported to have suffered for a week with a severe cold, for which domestic remedies had been employed.

On admission to the hospital, her temperature was 104° F.; her pulse, 130; her respirations, 70. Her color was somewhat dusky, and she was evidently greatly depressed by the fever and difficulty of breathing from which she suffered.

Upon physical examination, the lungs were found very much congested. Râles of different sorts were heard throughout the chest, while over the bases of the lungs tubular breathing was present.

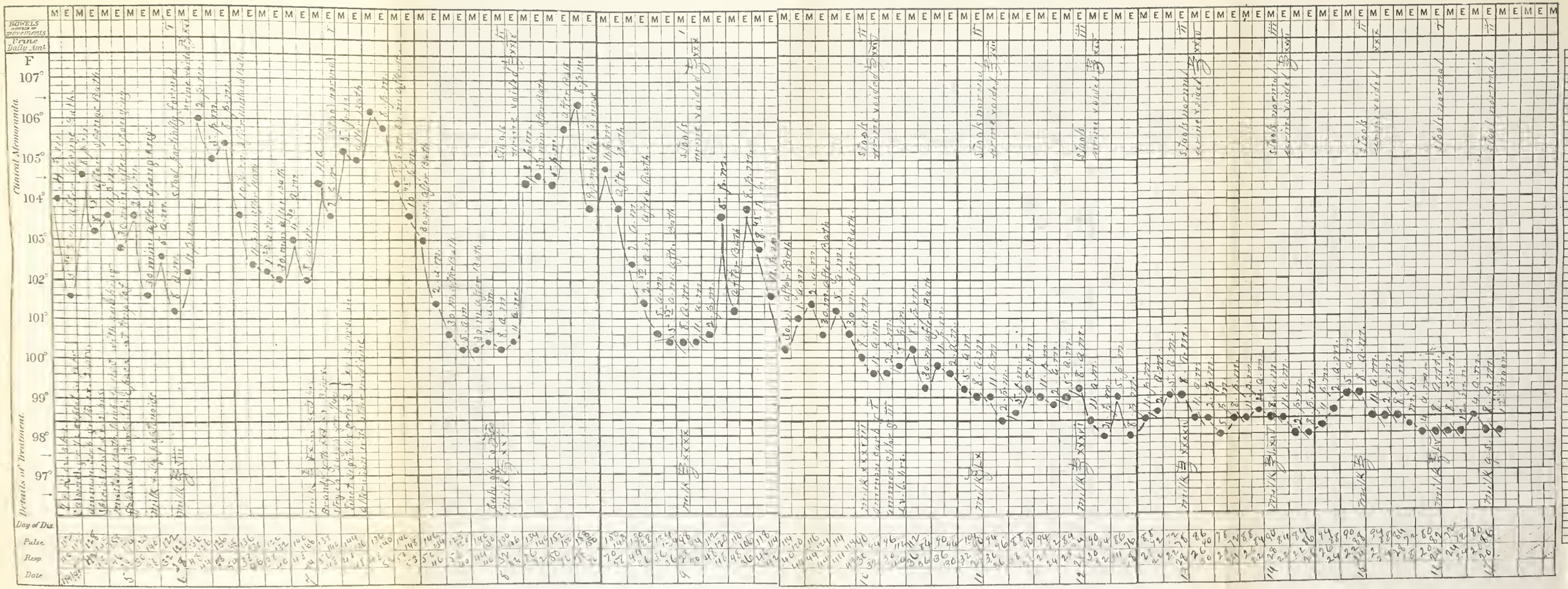
The child was, fortunately, able to retain food and stimulants. She was treated by the application of turpentine stupes to the chest, with the free administration of alcohol and preparations of ammonia and digitalis.

A temporary improvement followed, which lasted for thirty-six hours, after which her symptoms became greatly aggravated, and her temperature rose to 106° F. There was no marked cyanosis, and her symptoms resembled those of prostration from excessive temperature. She was sponged and packed in tepid and warm water to reduce fever. Warm baths were also employed, while stimulants were used freely, but without avail. Small doses of antipyrine had been used on one or two occasions, but with depressing effects. Failing to control the temperature, and the patient's depressing condition continuing, it was determined to try the use of cold. The patient was accordingly given a warm bath cooled down until the sensation to the hand was distinctly cold, and followed by the application of an ice-bag for a few moments to the lower and posterior portion of the chest. The resident physician was in readiness to administer stimulants hypodermatically, if signs of shock or collapse appeared. To the gratification of those who were caring for the child, not only was shock absent, but an immediate improvement followed the use of cold. Her temperature soon fell to 101°, her pulse improved, the respiration became more free, and the physical condition of the chest in a few hours showed decided improvement.

The subsequent progress of the case was uneventful to recovery.

It is evident that the occurrence of so high a temperature with its depressing effects should be considered as an emergency in the history of pneumonia among young children. It would certainly be difficult to attempt to treat a child in a private house by the application of cold, not only by reason of the lack of proper appliances and assistance, but also because of the seeming harshness of the method employed. There can be no question, however, that in the present instance the results were most satisfactory and striking.

In treating pneumonia in young children, it is well to remember the prominent part which congestion plays in the condition of the child, and in the danger to which the disease exposes it. In proportion as the lungs are excessively congested, collapse of the lung, or occlusion with mucus, with cyanosis, will follow. In treating this condition, digitalis and strychnia are of the greatest value internally. External applications are also of importance, and among them the most useful is the application of heat or cold. This is conveniently accomplished by the use of flannel wrung out of cold or hot water, sprinkled with turpentine, alcohol, or camphor, and wrapped about the entire chest. Over this is to be placed a dry flannel, and the whole is to remain upon the body of the child until it has become thoroughly dry. If stimulants are combined with the application of heat or cold, shock will not follow their use. In many cases the warm bath, to which mustard has been added, is most advantageous. In extreme cases, strychnia and atropia by injection are valuable. All of these patients, however, require stimulants and food from the beginning of the attack, given persistently and with careful judgment. Depressants are worse than useless, and should be avoided. While cold in the form of the bath or the ice-bag will seldom be required, in rare cases like that reported it may furnish the greatest stimulant available for the physician's use.


TEMPERATURE CHART IN PNEUMONIA.
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THE ASSOCIATION OF HEMIANOPSIA WITH CERTAIN SYMPTOM-GROUPS, CHIEFLY WITH REFERENCE TO THE DIAGNOSIS OF THE SITE OF THE LESION.

By CHARLES K. MILLS, M.D.,

AND

G. E. DE SCHWEINITZ, M.D.

Hemianopsias of intracranial origin are naturally first subdivided into (1) those due to lesions involving the optic tracts and primary optic centres; and (2) those due to lesions either of the visual cortex or of the internal capsule and subcortex.

Our remarks will be chiefly confined to strictly cerebral cases. These are of practical interest because of their comparatively frequent occurrence. They can be readily distinguished from the basal and peripheral cases; with less readiness, perhaps, but still as certainly, the subcortical can be separated from the cases of strictly cortical origin.

We have to present, first, a series of cases which, in their main features, correspond to a group of three cases described by Seguin,[1] in which lateral hemianopsia was one of a considerable group of what might be called hemisymptoms. Summarized, Seguin's three cases were as follows:

Case I. had a history of constitutional syphilis, and developed without an apoplectic attack, right hemiparesis, right hemianæsthesia and aphasia. Examination showed ataxic aphasia, alexia, with partial right-sided paralysis and anæthesia, and right lateral hemianopsia, the vertical line passing to the right of the point of fixation. His handwriting was awkward, and the hand and forearm exhibited, on voluntary effort, well-marked ataxic-choreic movements. Near the median line of the darkened half fields was a zone of imperfect vision.

Case II.—A man, aged sixty-two years, had an attack of hemiplegia and incomplete aphasia, with marked hemianæsthesia. He spoke much, but miscalled things. Vision was imperfect to the right. He improved greatly, but was left with very

[1] Jour. Nerv. and Ment. Dis., vol. xiii, No. 8, August, 1886, p. 446.

slight right-sided paresis, awkwardness, ataxic movements of the right hand and foot, marked tactile and ealoric anæsthesia of the right hand, loss of muscular sense, and incomplete homonymous (or lateral) hemianopsia, the right fields being darkened. Occasionally, he used a wrong word. He could see letters and numerals, but could not read except by a laborious process of spelling. Seguin regarded it as a blindness for words, or rather for the images or concepts which words represent.

Case III. was a man, aged twenty-six, with probable syphilis and history of apoplectic attack, from the first effects of which he decidedly improved. Examination showed partial right hemiparæsthesia and hemiparesis of the right upper extremity, with marked ataxia during volitional movements. He had right lateral hemianopsia, not quite reaching the point of fixation; about one-third of each visual field was obscured, and two-thirds of each half field.

Seguin regarded these cases as due to lesion of the outer edge of the thalamus, and of the internal capsule in its caudal part.

The five cases which follow correspond with those of Seguin in the presence of hemiparesis or hemiplegia; hemianæsthesia or hemiparæsthesia, partial or complete, and more or less persistent; speech-disturbances more complete at first, and later having the characteristics of an alexia or dyslexia, or a partial word-blindness; and lateral homonymous hemianopsia. The ataxic-choreic movements were not present, or more probably were not studied and described. In two of the cases (Cases I. and IV.), unilateral spasm was present.

The symptoms associated with the hemianopsia and speech disorder, sensory or motor, partial or complete, temporary or persistent, destructive or irritative, are distinct hemisymptoms, varying in degree, probably according to the radiation of the lesion from a common site.

Case I.—*Right Lateral Hemianopsia—Absence of Wernicke's Symptom—Dyslexia—Temporary Right Hemiparesis—Jacksonian Epilepsy.*—The first case has been already described by us in the proceedings of the Philadelphia Neurological Society.[1] We will, therefore, give the record here in a brief summary:

J. G. H., aged forty-five, had no history of syphilis, but had had two attacks of acute rheumatism. He noticed some failing sight and dizziness in the summer of 1888. December 17, 1888, he had an attack of left brachial monospasm, terminating with unconsciousness, and leaving him partially paralyzed in the right arm and leg, and with a marked affection of speech which lasted two or three weeks. In about three months, he had a second series of attacks of spasm, and after this he noticed he could see only half the objects to his right. He reads in a very peculiar

[1] Jour. Nerv. and Ment. Dis., vol. xv, No. 1, January, 1890, p. 55.

way—slowly, and pronouncing each word separately, or at most two or three words; he seems to have difficulty in seeing the word; he says that he sees it plainly, but that he soon gets mixed and confused. He can write a few words, and then his hands fail to inscribe further letters, while his face gives evidence of chagrin at the abortive attempt. Occasionally, when walking in the street, he imagines he sees something that does not exist—always to the right. Hearing and touch are normal.

The following notes by Dr. de Schweinitz describe his ocular condition:

In the right eye the sharpness of sight was equivalent to $\frac{2}{7}$ of normal; in the left eye $\frac{2}{5}$ of normal. This deficiency in visual acuity was probably due to the presence of a mixed astigmatism.

In the right eye the optic disc was a vertical oval, bounded at its outer margin by a black line, its surface a little woolly, and all the capillaries injected; the edges of the disc, however, were not obscured.

In the left eye the disc was distinctly gray in color, its hue being manifest through a superficial injection of the surface capillaries. The temporal half of the disc was unobscured; the nasal edges slightly blurred. In neither eye was the disc swollen, nor were there any splotches or hemorrhages in the retina.

The pupils of both eyes were equal in size, and reacted normally to the changes of light and shade, convergence and accommodation. The hemiopic pupillary inaction (Wernicke's symptom) was not present in either eye.

There was complete right lateral hemianopsia, the field of the left side being proportionally much smaller than its fellow on the right, and both the preserved fields exhibiting concentric contraction. The dividing line on the left side almost cut the fixing point. That on the right side, on the horizontal meridian, touched the fixing point, while above and below this it spread five degrees from the centre, making a curious re-entering angle.

CASE II.—*Right Lateral Quadrant Anopsia—Absence of Wernicke's Symptom—Dyslexia—Right Hemiparesis—Partial Right Hemianæsthesia—Partial Word-Deafness and Word-Blindness.*

J. B., aged forty-two, a laborer, May 3, 1892, had an apoplectic attack with loss of speech, and was taken to the Hospital of the University of Pennsylvania, where he remained until November, 1892, when he came to the Philadelphia Hospital. He improved slowly. During his stay in the University Hospital, he noticed when he grasped an object with his right hand that he could not release it as promptly as when he seized it with his left.

Examination showed that he was paretic in the right arm, and his field of vision towards the right was decreased. He says that at times he could not understand what was said to him, especially if the person speaking stood to his right. Examination showed paresis of the right forearm, partial right hemianæsthesia, tongue slightly tremulous on protrusion, pointed and deviating a little to the right. He had gradually recovered his speech.

From the first he says he knew what he wanted to say, but could not put it into

such words as would be understood by his hearers. He could recognize objects, but could not pronounce their names. He could not read writing or print because everything ran together; he recognized the letters, but could not pronounce them. He understood sentences, but could not repeat them. He had almost completely recovered from his aphasia, although his voice was a little thick and difficult to understand. One of the most marked phenomena was that which has been described as dyslexia.

The examination of his eyes gave the following results:

R. E.—Oval disc, shallow, dish-like excavation, almost complete. Vessels about normal in size; no fundus lesions.

L. E.—Small, nearly round disc, and small physiological cup. Vessels almost normal in size.

Pupils equal and round; mobility of iris normal; no hemiopic pupillary inaction.

Fields.—Right lateral quadrant anopsia. (See Figs. 1. and 2.)

FIG. 1.

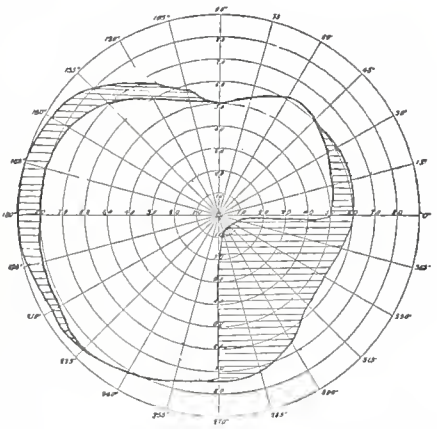
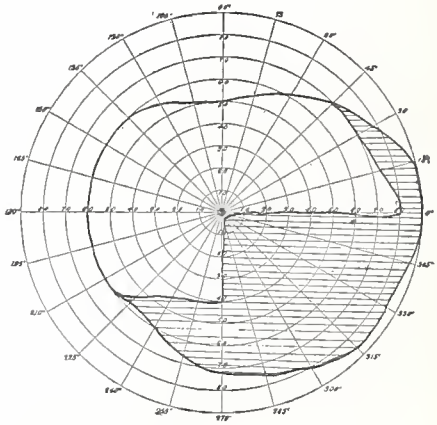


FIG. 2.



Visual fields of Case II. Right quadrant anopsia.

CASE III.—*Right Lateral Hemianopsia—Absence of Wernicke's Symptom—Temporary Aphasia—Dyslexia—Right Hemiparesis of Spastic Type—Probable Word-Blindness.*

W. V., aged fifty-four; in November, 1889, first noticed that when seizing anything with his right hand he could not let it go as soon as he wished. Soon the arm and leg of the same side showed a tendency to rigidity, and became in a month and a half so paretic and spastic that he was unable to walk.

He also, about the same time, became completely aphasic; but he understood everything that was said to him. He was apparently word-blind but not mind-blind. He said of himself: "They showed me things, and I knew what they were, but could not pronounce them." At the end of about three months his speech began to improve.

When examined in November, 1892, he was found to have spastic right-sided hemiparesis without anæsthesia. His tongue on protrusion was slightly tremulous. He recognized objects but hesitated when asked for names, which he could some-

times give. He reads with difficulty, dyslexia being marked, and of much the same character as shown in Case I.

Examination of his eyes by Dr. de Schweinitz gave the following results:

R. E.—Oval disc, faint superficial capillarity, but deeper layers gray. Vessels fuller than usual, arteries contracted; slight epithelial choroiditis.

L. E.—Similar optic disc, but much more gray and both sets of vessels contracted.

Pupils round, 3 mm. in diameter, acting equally and normally to convergence, accommodation and light stimulus; hemiopic pupillary inaction wanting.

Fields.—Typical right lateral hemianopsia, with contraction of the preserved fields. (See Figs. 3 and 4.)

FIG. 3.

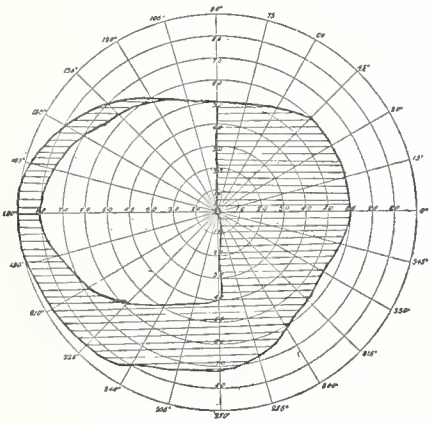
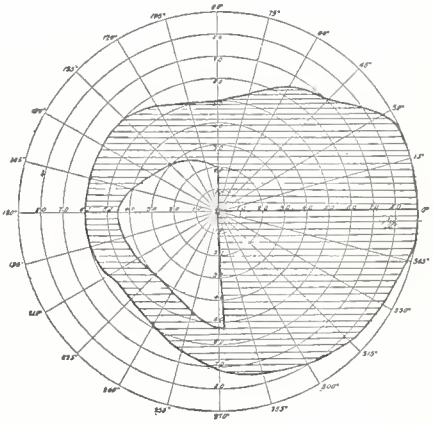


FIG. 4.



Visual fields of Case III. Right lateral hemianopsia.

The symptom described as dyslexia is one of interest, both from the clinical standpoint and in the view of the assistance which it may afford in locating the lesion. Dyslexia, as defined by Swanzy, consists in a want of power on the patient's part to read more than a few words consecutively, either aloud or to himself. Post-mortems have been made in several cases of dyslexia reported by Berlin, and in one recorded by Nieden. The lesions are said to have been found in the white matter near Broca's convolution. Such symptoms would, on general principles, be attributable to partial destruction of the entire centre for word-images, or of the association-fibres which connect this centre with the emissive regions for speech and writing. Most probably the lesion would be an incomplete destruction of commissural-fibres, and hence, subcortical.

CASE IV.—*Left Lateral Hemianopsia—Left Hemiplegia and Hemianæsthesia—Partial Aphasia, Temporary—Epilepsy—Absence of Wernicke's Symptom.*[1]

M. B., aged twenty-eight, after the birth of her last baby had a post-partum hemorrhage, also probably thrombosis of the left femoral vein, from the effects of which she was three months recovering. Three weeks after her labor, she had an apoplectic stroke, with unconsciousness. She was left with hemiplegia and hemianæsthesia of the left side, and with some thickness of speech, which remained for a month. Two weeks after the stroke, she had for the first time a fit, in which she was unconscious, and her entire left side was convulsed; she had a series of convulsions in a period of five hours. She bit her tongue and frothed at the mouth. She has since had, from time to time, attacks of spasm, which begin with vomiting and auditory and visual aura; she has hallucinations of hearing voices, and also has hallucinations of sight; she says that she sees diamonds sparkling.

Examination of her eyes showed disseminated choroiditis and left lateral hemianopsia without Wernicke's symptom.

CASE V.—*Right Lateral Hemianopsia—Absence of Wernicke's Symptom—Temporary Right Hemiplegia—Persistent Hemiparæsthesia.*

G. W. R., aged forty-five; patient of Dr. H. C. Wood; examined by Dr. de Schweinitz October 12, 1889. About a year ago she suffered from right-sided

FIG. 5.

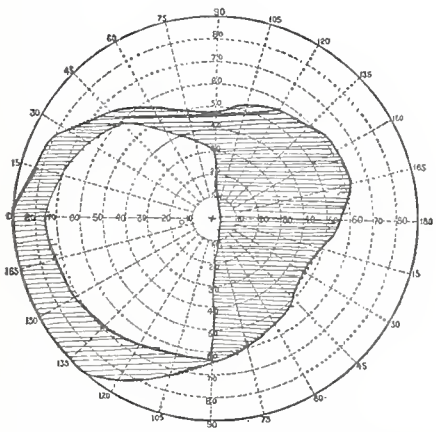
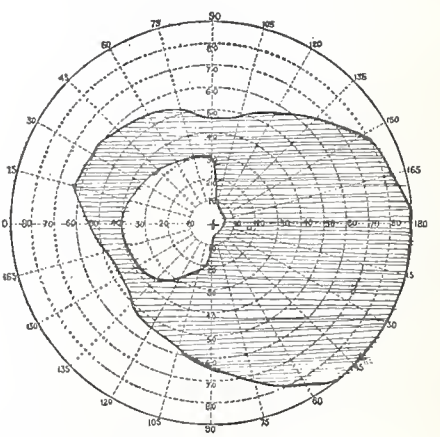


FIG. 6.



Visual fields of Case V. Right lateral hemianopsia.

hemiplegia, which seems to have been temporary, and has been followed by a persistent numbness of that side. Unable to read or use her eyes. Urine contains albumin.

Vision with correcting glass one-half of normal. In the right eye a gray, oval optic disc, with choroidal changes below it of a streak-like character, and fine dot-like changes in the choroid of the macular region; general absorption of the pigment epithelium.

[1] This case was observed in the service of Dr. S. Weir Mitchell, at the Orthopedic Hospital and Infirmary for Nervous Diseases.

In the left eye an irregularly oval disc, gray, and disseminated choroidal changes, especially down and in and below. No changes in the external ocular muscles; Wernicke's symptom absent.

Fields.—Typical right lateral hemianopsia, with contraction of the preserved field, most marked upon the right side. (See Figs. 5 and 6.)

In the next two cases the hemisymptoms are slight and ill-defined, but the hemianopsia is complete and regular, indicating lesions of the optic radiations close to the internal capsule.

CASE VI.—*Right Lateral Hemianopsia—Right Hemiparæsthesia.*[1]

J. P., aged fifty-six, machinist. Seven months before coming under observation, complained of general sensation of numbness on the right side of the body, which the doctor called "la grippe." He also was weak on this side, and found that he could not read. The right-sided numbness has continued until the present time. His tongue is protruded tremulously, and is perhaps slightly pushed to the right. He has been much troubled with vertigo. He admits having gonorrhœa, but denies having syphilis. He has sometimes smoked as many as 200 cigars in a week. Otherwise, nothing special in his history or in the results of the examination, except in visual phenomena.

Dr. de Schweinitz found that he had right lateral hemianopsia without Wernicke's symptom, and with no disturbance of the fundus, and no color-blindness or deficiency in color-sense.

CASE VII.—*Right Lateral Hemianopsia—Absence of Wernicke's Symptom—Doubtful Right Slight Hemiparesis.*

J. S., private patient of Dr. de Schweinitz, a woman, aged fifty, examined November 2, 1891. She was in generally fair health without any severe illness until one year ago, when she suffered from a fever, probably typhoid. Since that time has complained of weakness, and especially of imperfection in vision, being confused when she is walking, particularly going up and down stairs. She believes she has suffered thus for two years, and has not been well since the climacteric, which took place at about that time, a statement that is confirmed by her family physician. There is slight lameness of the right leg, which is somewhat favored in walking. This lameness is said to have improved under anti-rheumatic treatment. There is no anæsthesia, no change in the muscle or knee-jerk, and no disturbance of intellect. There has never been any unconsciousness, convulsions or cerebral symptoms of any type, not even headache.

Central vision, after the correction of a myopic astigmatism, is normal. The external ocular muscles are natural in their action, there being only a slight insufficiency of the internal recti. Each optic disc is a vertical oval, gray in its deeper layers, with some massing of pigment on the nasal side. The retinal vessels are normal in size and carry normally tinted blood. The pupillary reactions are natural, the contraction to light being present, no matter upon which side of the retina the beam is thrown.

[1] This patient was observed in the service of Dr. Wharton Sinkler, at the Orthopedic Hospital and Infirmary for Nervous Diseases.

There is right lateral hemianopsia with much contraction of the preserved fields, the dividing line making a re-entering angle at the fixing point. The field of vision was tested with a piece of white, 1 cm. square. (See Figs. 7 and 8.)

FIG. 7.

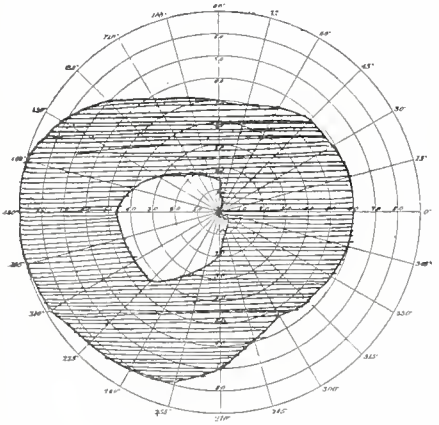
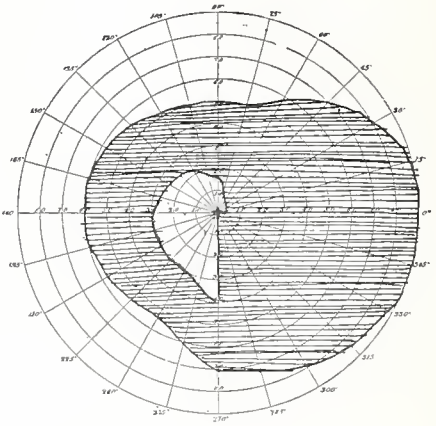


FIG. 8.



Visual fields of Case VII. Right lateral hemianopsia.

CASE VIII.—*Left Lateral Hemianopsia—Absence of Wernicke's Symptom—At First, Paresis of Both Legs—Later, Right Spastic Crural Monoparesis.*[1]

In January, 1892, the patient made an unsuccessful attempt to commit suicide with "Rough on Rats." The next morning he shot himself in the head three times. According to his own statements, twice pointing the pistol to the right parietal region and the third time to the top of the head. He lost consciousness and knew nothing of what was going on for three weeks. When he attempted to walk he found that both legs were partially paralyzed, or at least very weak, the right being more affected than the left. He was also weak and shaky in the arms, but thought that he had no paralysis of the upper extremities. He was told that he had had five or six convulsions while he was unconscious, but he had had none since getting on his feet.

Examination showed spastic paresis of the right leg with exaggerated knee-jerk and muscle-jerk on the right; no anæsthesia. He has a scar in the median line about 9½ inches back from the glabella; another about 11½ inches back, and a third scar and apparent depression about 2½ inches to the right of the median line, between the positions of the other two scars. The most posterior scar is in about the position of the occipital protuberance; and that to the right of the median line is over the position of the occipital lobe.

From the first this patient has complained of greatly impaired vision. Examination showed left lateral hemianopsia, with absence of Wernicke's symptom, and no changes in the fundus or media. (See Figs. 9 and 10.)

[1] This man was a patient in the service of Dr. S. Weir Mitchell, at the Orthopedic Hospital and Infirmary for Nervous Diseases, and later in the Polyclinic and Philadelphia Hospitals, service of Dr. Mills; he was, in fact, something of a hospital rounder, having been in various hands in different cities.

In this case the hemianopsia is to the left, the marked spastic paresis to the right. The most probable explanation would seem to be wounding of the right cuneus and cerebral subcortex, and by the same bullet (?) of the motor tract (by fibres) on the left.

It is difficult to distinguish clinically between cortical hemianopsia and that due to limited lesion of the optic radiations in the occipital

FIG. 9.

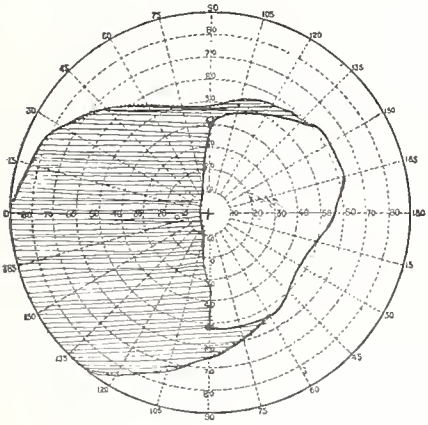
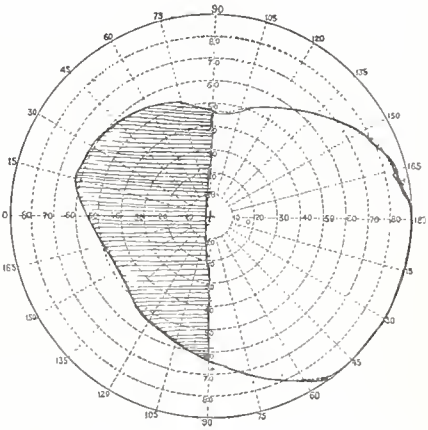


FIG. 10.



Visual fields of Case VIII. Left lateral hemianopsia.

subcortex; but in the latter the hemianopsia is likely to be more typical and regular in character.

The following cases are probably *cortical:*

CASE IX.—*Atypical Left Lateral Hemianopsia.*

J. B., private patient of Dr. de Schweinitz, aged eighty-three, examined December 15, 1890. Has always been a very healthy man, but for three weeks has com-

FIG. 11.

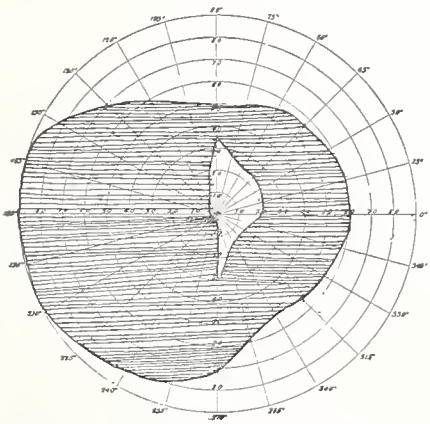
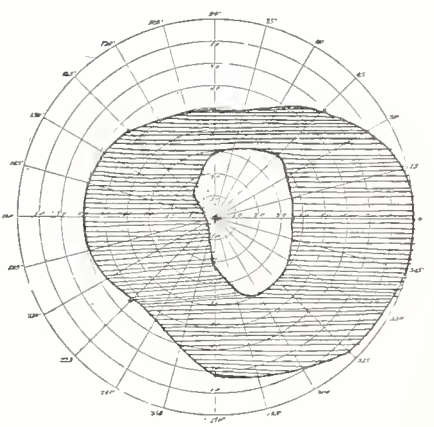


FIG. 12.



Visual fields of Case IX. Left lateral hemianopsia, with marked contraction of left half-field.

plained of failure of sight; no pain; occasional attacks of rheumatism; arteries atheromatous everywhere; urine contains albumin, hyaline and granular tube casts. Vision, 10/C; pupillary reactions sluggish but normal.

Eyegrounds present in moderate degree the appearance of a degenerative type of albuminuric retinitis, the spots of fatty degeneration and fine hemorrhages being chiefly confined to the macula. Few changes occurred, except a slow decrease in the visual power and a moderate increase in the retinitis, indicated chiefly by the appearance of fresh flecks of hemorrhage and the beading of the upper nasal vein.

July 3, 1891, while standing in the sun, was suddenly attacked with a giddy spell, pain in the back of the head and the back, and confusion of ideas. This soon passed away, and he was able to go home unassisted.

The field of vision on the following day presented the character in the diagram, namely, atypical left lateral hemianopsia, the dividing line being somewhat in advance of the fixing point. (See Figs. 11 and 12.) He continued to live as late as December 25, 1891, having had a number of attacks precisely like the one described. After this he was not seen.

CASE X.—*Right Lateral Hemianopsia—Absence of Wernicke's Symptom.*

S. P., a private patient of Dr. de Schweinitz, aged sixty-four, examined December 18, 1891. For six weeks has suffered from subjective and objective vertigo; loses place in reading; some dislocation of thought processes, and fails at times to connect words and sentences. The feeling is described as that of a man who has taken too much wine. There is some loss of sleep, and a great deal of headache, chiefly frontal, and rather more to the right side than the left. This headache is now almost constant, and is described as a pressure rather than a pain.

When examined there was no paralysis, no apparent disturbance of intellect, and, indeed, no marked symptom except the vertigo and the headache.

Central vision normal after the correction of a myopic astigmatism. No palsy of the external ocular muscles. Exophoria, two degrees at twenty feet. Pupillary reactions entirely normal. In the right eye an oval optic disc, gray-red, the nasal edges veiled, and a stripe of glistening tissue across its lower margin. No change in the central vessels. In the left eye a round disc, distinctly gray, the scleral ring too well-marked on the temporal side, but the nasal edges veiled.

Typical right lateral hemianopsia with moderate contraction of the preserved field, the contraction being relatively greater upon the right side. The central color perception was normal. The field of vision was taken with a square of white, 1 cm. in diameter. Three days later the patient died suddenly, with the clinical symptoms of a large apoplexy.

Certain broad distinctions can be made between cortical hemianopsia and those subcortical cases in which the lesion involves both the optic nerve and the internal capsule. In these the hemianopsia is associated with certain definite hemisymptoms, such as were present in the majority of cases here enumerated. The symptoms may vary considerably within certain limits. Sometimes both hemiplegia and hemi-

anæsthesia are present. Usually these conditions are partial; frequently one is present and the other is absent. Other hemisymptoms of somewhat frequent occurrence are ataxic chorea and monospasm or unilateral convulsions.

In some cases the paresis or paralysis is decidedly of a spastic type. In this group of cases the lesion is probably located at a position where the commissural-fibres, the optic radiations and the internal capsule come together, probably at a point in the centrum ovale near the junction of the parietal and occipital lobes. We do not believe that the thalamus is usually involved in these cases, although it may be in some instances. While we have no pathological disproof of this, we have evidence of a negative character in a marked case of hemianæsthesia of many years' standing, without hemianopsia, and in which the autopsy showed a large lesion of the thalamus, with moderate involvement of the internal capsule.

Double hemianopsia is rare, about thirteen cases being on record. These cases throw light on macular representation. Through the kindness of Dr. T. D. Dunn we have been enabled to study one case of this character, and we reproduce Dr. Dunn's report from the "University Magazine," May, 1895:

DOUBLE HEMIPLEGIA WITH DOUBLE HEMIANOPSIA AND LOSS OF GEOGRAPHICAL CENTRE.[1]

Dr. Dunn writes thus:

"The case which especially attracted my attention to the subject treated in this paper is a man of sixty-eight years, married, who has led an active life. His parents lived to be nearly seventy years old. Four or five older brothers and sisters, and three of his four children are living and healthy. He is not addicted to the use of alcoholic drinks, and there is no history of syphilis. He fancied a good horse, and rode and drove much.

"In 1888 he was thrown from his carriage and received several severe scalp wounds, but no fracture. He was unconscious for several hours. From this accident he completely recovered, although he was very nervous for several months.

"During the winter of 1890, while in Philadelphia, he had an attack of epidemic influenza, followed by pneumonia, and was then under the care of Dr. H. C. Wood. With the exception of the above, he had had no severe illness, though he frequently applied to me for attacks of acute indigestion, which always brought on violent cardiac palpitation. The pulse would become intermittent, irregular, and often 130 per minute. These attacks seldom lasted more than two days, and during the intervals the heart's action was fair. He had no dyspnœa, though there was

[1] Read by Dr. Dunn before the College of Physicians of Philadelphia, March 6, 1895.

mitral valvular disease with compensatory hypertrophy and general arterial hardening.

"On the morning of May 2, 1891, he came to my office with one of these attacks. Being more comfortable in a few hours, he took a drive of eight miles to dine with a country friend. He ate a hearty dinner, and shortly afterwards became unconscious.

"At 8 P. M., two hours after the attack, I found there was partial paralysis of the right leg, nearly complete paralysis of the right arm, slight drawing of face, and partial right hemianæsthesia. He was unable to articulate any words except 'yes' and 'no.' Cold applications to the head and a calomel purge were prescribed.

"The following day the movements of both arm and leg were improved, but he complained bitterly of pain in the head and eyes. Much relief was obtained from bromide and cups to the back of the neck.

"On the third day right lateral homonymous hemianopsia was discovered, which doubtlessly dated from the beginning of the attack. He recognized persons and objects, but could not name them; movement of the right side was much improved, and he complained less of numbness.

"At the end of ten days the hemiplegia had nearly disappeared, the facial expression was normal, and the hemianæsthesia scarcely perceptible; knee-jerks increased. His mind was clear and there was considerable increase in his vocabulary, but no improvement in the field of vision.

"In three weeks he was walking about as usual, and his general condition was as good as before the attack. Vision remained unchanged, and there was a slight tendency to bear to the left in walking. The aphasia slowly improved, though much trouble was found in recalling the names of things. With the exception of an occasional attack of indigestion, he enjoyed good health, and drove and rode as usual, though his use of names was never entirely restored, and there was no increase in the visual field.

"Dr. G. E. de Schweinitz examined the patient's eyes November 24, 1891, and supplied the following report:

"'*Vision* of the right eye $\frac{20}{XL'}$, of the left eye $\frac{20}{XXX}$. With +3 D, 0.50 m. was read with each eye at thirty centimetres. The excursion of the eyes was good in all directions; there was no paresis of any of the external ocular muscle, no history of diplopia, and only slight insufficiency of the internal recti muscles with the ordinary tests.

"'*Ophthalmoscope—Right Eye.*—An irregularly oval optic disc, gray in its deeper layers, and bounded by a pigment crescent at the outer side; veins full and tortuous, arteries normal; general fundus in good condition and without signs of former disease.

"'*Left Eye.*—Irregularly oval, slightly gray, optic disc, edges a trifle prominent but clearly outlined; veins full, arteries normal; no abnormality in the retina or choroid.

"'*Field of Vision.*—Typical right lateral hemianopsia with contraction of the preserved fields, the relatively smaller half field being upon the right side. The dividing line between the preserved and the darkened fields passed, as is usual, slightly in advance of the fixing points. (Figs. 13 and 14.) Central color perception was normal.

"'*Pupils.*—Contraction of the iris to light was present equally when the beam

was thrown upon the blind and upon the seeing side of each retina. In other words, the hemianopic pupillary inaction was wanting, consequently the lesion which determined the hemianopsia was situated posterior to the primary optic centres. The pupils were round, equal in size, and the iris reactions normal in all other respects.'

"January 28, 1893, after eating imprudently, he had an attack of indigestion with the usual cardiac disturbance, and awakened the following morning with left hemiplegia and partial hemianæsthesia, associated with absolute blindness. Speech was not affected, but he was much worried over the loss of power in the left side and loss of sight, and he had no conception of where he was. Taste and smell were natural, voices were recognized, and his mind was only slightly affected.

"Four days afterwards, as after the first attack, there was rapid restoration of power and sensibility, but no improvement in vision or knowledge of locality. His mind was clear, and he would discuss his condition intelligently.

FIG. 13.

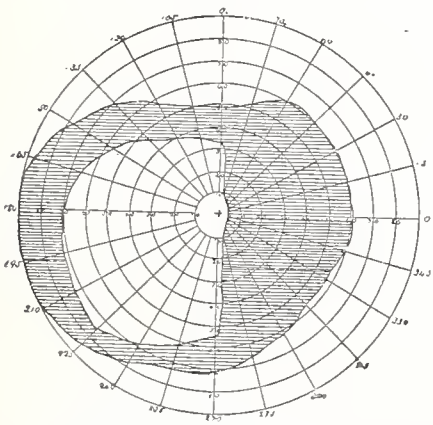
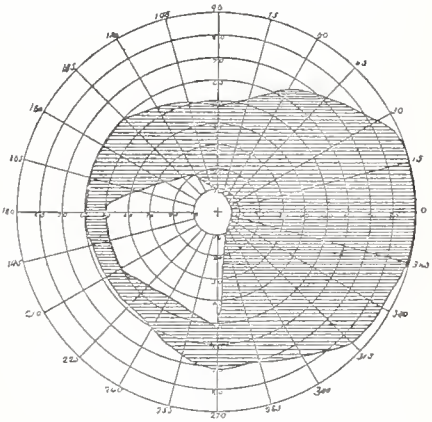


FIG. 14.



The external continuous line marks the boundary of the normal visual field, the internal continuous line the limits of the field in the case examined, the white area the region of preserved vision, the shaded area where vision was lost.

"On the eighth day he recognized light from a window, and when a candle was placed directly in front of him he saw it with each eye, but nothing could be distinguished in the room, and he could not recognize individuals except by their voices. He had much trouble in locating himself and his house—being unable to form a mental picture of any place.

"At the end of four weeks power and sensibility were restored, and he could walk, when led, as well as any blind man.

"With his reading glasses letters could be recognized at his usual reading distance, but only the smallest words could be seen without changing the position of his head. Of the word 'constantly,' in ordinary reading type, he could see six letters, but not the remaining four. He could, however, read slowly by taking in small words and parts of large ones. There was no improvement in the sense of locality. He could form no conception of the geography of his own house or of any place he had ever been. He could recollect that he lived on the corner of two streets and their names, but their relation to each other, or to other streets, was completety lost. He could not see enough of the sketches made of the floors of his own house to assist him in getting an idea of the relation of things. When a

place was named he could remember it, but its location or relation to other places was lost. He could recollect individuals by their voices, and could describe correctly their appearance.

"Dr. de Schweinitz examined the patient's eyes again April 19, 1893, when the following conditions were noted:

"'*Vision* of the right eye equalled one-half, that of the left also one-half of normal. With + 3 D ordinary newspaper print (pica and small pica) could be read very slowly, one word at a time, by holding the head in such position that the word corresponded exactly with the point of fixation. If the word was long, only a portion of it could be read at a time.

"'*Ophthalmoscope.*—The appearances of the optic nerve, bloodvessels and general fundus of each eye were the same as those noted in the previous examination, save only that the grayness in the deeper layers of the discs was more manifest, and there was slight blurring of the nasal edges of the nerve-heads.

"'*Field of Vision.*—The visual field in each eye was obliterated, with the exception of a small, irregularly oval patch directly in its centre, about ten degrees in the horizontal and slightly greater in the vertical diameter. In other words, there had been added to the previous condition a left lateral hemianopsia. The preservation of this small central field in each eye indicated that the region in the cortical visual centres which supplies the macula lutea had not been destroyed—another clinical demonstration of the special representation of the macula lutea in the cortex. (Figs. 15 and 16.)

FIG. 15.

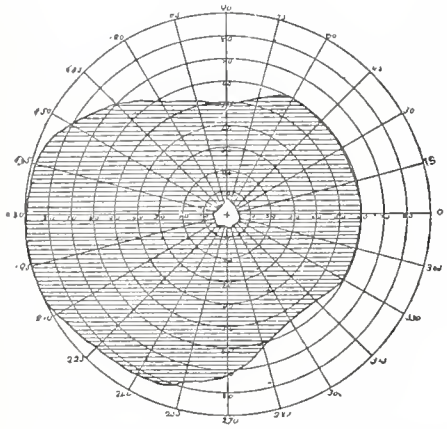
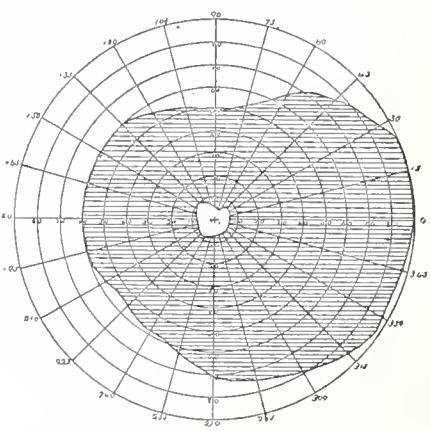


FIG. 16.



Diagrams of the visual fields after the second attack of hemianopsia. The small central white area represents the limits of the preserved field (macular vision); the shading, where vision was lost.

"'*Pupils.*—The iris reaction, when a beam of light was directed into the pupillary space so as to fall, if possible, on the macular region, was normal in each eye, although the response was less prompt or almost absent when the beam was thrown to either side of this position.

"'*Color-sense.*—This was practically lost; red gave the sensation of 'brass,' green that of 'gray-white,' yellow was called 'gold but not bright enough,' and blue was described as a color that 'might be purple.'"

"In January, 1895, he had another attack of pneumonia, not extensive, from which he recovered, but with weakened heart action and considerable dyspnœa.

"It is now (March 1, 1895) over two years since the second attack of hemiplegia. There is no perceptible improvement in the field of vision; there is a small central point which can recognize a pin on the floor, but he can see only short words at ordinary reading distance. Although he has driven over the town and walked about with an attendant almost daily, nothing has been gained of the lost sense of locality. Two points show slight improvement in this respect in his own home.

"After coming from a walk, if he is led to the front door and stands face inward, he can walk to a smoking-room at the opposite end of the thirty-foot hall; if, however, his face is turned outward, or even a quarter around, he cannot find his way. In his sitting-room, he fixes his position by the tick of the clock and can find his way out of one door to a bath-room, or out of another to the hall, where the banister enables him to descend the stairs to the dining-room. The aphasia has nearly disappeared, though some words are still recalled with difficulty.

"His general condition is fair, and he discusses his case with much interest and intelligence."[1]

A very few cases have been put on record in which one-half or a portion of the macular field has been affected.

We have notes, somewhat imperfect, of an unpublished case which would seem to teach us that we may have either functional or organic disturbance of the word-symbol centre of the same character as that which produces hemianopsia when the half centre for general vision is destroyed on one side of the brain.

This patient was an educated man, engaged in scientific work, and had been troubled for many years with flying specks and one dark spot. When sitting, the flying bodies went downward from above; turning his head from side to side, they fell to either side, according to the direction in which his head was moved. On closing his eyes he often had phosphenes—a multitude of little, bright bodies, like blood-corpuscles in size, upon an orange field. He was highly hyperopic, but did not wear spectacles until he was forty years old. He had been a close student, frequently using the microscope.

The peculiar half vision with which he suffered at times consisted in the right half of a word disappearing or becoming blank while he was reading. For example: in the word "capacity," the letters "acity" would suddenly be blotted out. All around the obliterated letters he could see words and objects, but indistinctly. The trouble was the same with both eyes open or with either eye shut; the right half of the word was blotted out for the right eye, the left eye for both. By twisting or moving his head, so as to bring his eyes along to the right, he could pursue his reading, the blank space receding towards the right. (Noyes.)

[1] Since this was written the patient has died; autopsy was refused.

TWO CASES OF CEREBELLAR TUMOR.

By WHARTON SINKLER, M.D.

Tumors of the cerebellum are comparatively rare. So few cases have been reported in which the diagnosis was made before death, and confirmed by post-mortem examination, that the following cases are worth putting on record.

The symptoms of cerebellar disease are more or less uniform, although the character of the symptoms depends largely upon the region of the cerebellum involved. Unsteadiness of gait and a tendency to fall to one side or the other, are generally met with in cerebellar disease, and changes in the eye-ground are almost always encountered early. The ocular symptoms, however, are not invariable, for we occasionally meet with cases in which the fundus of the eye is apparently normal for a long time after the development of a growth. Frequent examinations of the eye should be made, for often optic neuritis may develop suddenly. Loss of vision following optic neuritis is an early symptom in cerebellar disease. In Case II., the failure of vision first brought the patient under notice. Nystagmus is a symptom common in cerebellar disease, but it was not present in Case I. Speech defects are also often present, the scanning speech, like that which occurs in disseminated sclerosis, being the form of defect usually observed. In some cases the differential diagnosis between cerebellar tumor and disseminated sclerosis is difficult to make, for in both we may have the unsteadiness of gait, nystagmus, scanning speech, and inco-ordination in the movements of the upper extremities. In both of the cases which I shall report the diagnosis of cerebellar tumor was made before death. The main points in making the diagnosis in Case I. were the occipital pain, peculiar unsteadiness of gait, cerebellar titubation, optic atrophy and vomiting; and in Case II., the occipital and post-cervical pain and optic atrophy. An interesting clinical point in Case II. was the immediate relief of the violent occipital pain which was afforded by leeching.

CASE I.—R. S., aged forty, born in England, a sailor by occupation. Admitted to the Philadelphia Hospital March 10, 1892. He states that he has enjoyed good health, but that several years ago, while bathing, he received a violent blow on the occiput. No clear or distinct history can be obtained from him, for his statements are usually exaggerated. Some time in June, 1891, he noticed a stiffness in the fingers of the right hand and pains in the head, for which he took large quantities of bromo-caffein. He asserts that he had had attacks of nausea in the morning. His statements are all extravagant. He declares that he can speak German, Danish, Swedish, Norwegian and Dutch as well as his native tongue. He also maintains that in 1877 he walked 3700 miles along the coast of India, and that he frequently has walked from Providence to Boston. He made other equally exaggerated statements.

A letter from a well-known firm in Camden, in reply to an inquiry made by the resident physician, gave us information in regard to this patient which was somewhat different from his own statement. It seems that the man had been going to the offices of the company, both in Camden and New York, for six or eight years, buying pens, which he peddled about the country. The writer stated that in the early part of his knowledge of the patient he appeared quite strong physically, but was always peculiar mentally. "He used to speak of himself, and we thought truthfully, as a crank. His mental peculiarities appeared to increase within a year. He appeared here in the late winter and early spring, 1892, complaining of giddiness and continued disposition to staggering, and giving evidence of increased weakness mentally."

On admission he could walk without support, but staggered from one side to another like a drunken man; the reflexes were increased, sensation was normal. There was ptosis of right lid, and the right pupil was smaller than the left. An ophthalmoscopic examination shows no advanced change in the discs. He could read at the normal distance.

March 28, 1892.—Gait is more staggering and the patient is weak; station is unsteady; there is ataxia of the hands and fingers, but no tremor. There is a large lump on the back of the head, right of the posterior occipital protuberance, about the size of a peach. It is soft, painless and movable.

April 7, 1892.—The patient falls to the right when he attempts to walk, and now has to be tied to his chair to prevent him from falling from it, his body bending to the right side as he sits. The appetite continues good. There is now incontinence of urine. His mental condition continues about the same. He is at times noisy, but generally is happy and has delusions of grandeur. Speaks of having large sums of money in his chest, which he says he left at a boarding-house near the river front.

April 18, 1892.—On account of increasing weakness, has been obliged to remain in bed. Cannot feed himself, and there is incontinence of urine and fæces.

April 26, 1892.—He now vomits his food without apparent nausea. There is increasing hebetude.

An ophthalmoscopic examination was made on April 10th by Dr. Gould, and the following is his report: "The fundus oculi shows progressive atrophy, the arteries being lessened in size to a mere thread; the temporal sides of the discs show more decided atrophic change. The veins are also decreased in size, but not so much as the arteries. The left optic disc is very oval, and is of an unsymmetrical axis. The fundi are congested and unhealthy in appearance. There is

progressive ptosis of the right lid, but no other muscles supplying the eye are affected. The papillary reactions are very sluggish."

The patient gradually became weaker, with constant increasing hebetude, and died June 23, 1892.

The *post-mortem examination* was made by Dr. John Guitéras. No abnormalities were discovered in the thorax or abdominal cavity, with the exception of a small abscess in the spleen and a small cyst in the left kidney.

Head.—Nothing abnormal in the skull, except that the cranial vault is thickened and dense, especially in the posterior portion; rather flaccid vessels, not distended; membranes somewhat opaque; the membranes below the longitudinal sinus present nothing abnormal. Upon removing the dura mater, the pia mater appears somewhat opaque for a distance of about three inches from the median line. The opaque portions of the pia show, especially along the course of the veins, numerous minute white tubercular bodies. At the base of the brain the pia is generally normal, except that the same opacity is seen along the Sylvian fissure. The bloodvessels at the base of the brain are normal. There is a fungoid mass in the pia covering portions of the pons, medulla and cerebellum of the left side. It is two and three-quarter inches long by two and one-quarter inches wide. It extends along the anterior portion of the cerebellum to the pons and left peduncle. The latter structure is partially covered. It covers the pons to within one-half inch of the median line, and extends backwards, approaching the median line, so that the whole left half of the medulla is covered by the mass. The posterior portion of the basillar artery is also involved. All the nerves of the left side, from the fourth back, pass out through the mass. The mass can readily be lifted from the medulla and pons, being adherent only to the cerebellum, from which it appears to spring at the lateral course between the inferior and superior surfaces and the cerebellum. The cortical pia peels off very readily from the brain, but the brain is somewhat thickened and dotted with the white miliary granules already mentioned.

Brain.—The lateral ventricles are somewhat dilated, and contain a considerable amount of fluid. On cutting through the middle lobe of the cerebellum from above along the median line, a tumor was exposed, which filled the fourth ventricle. The cavity of the ventricle is decidedly distended by the mass. The latter extends across the median line into the right side, and eventually penetrates the ventricle from the left side. It can be readily lifted from the right side of the floor of the ventricle, but it is attached to the left half of the floor up to a short distance from the median line. On the right side the floor presented a normal appearance. The mass in the ventricle is about one and one-quarter inches long, and three-quarters of an inch wide.

The tumor involves the gray matter of the cerebellum, affecting the tonsils, the inferior middle lobe, and the flocculus. It is well circumscribed in the front by the great horizontal fissure, and posteriorly by the inferior posterior fissure. Beyond this area the tumor extends upon the surface of the cerebellum, pons and crus, upon all of which it can be easily separated, except at the area above described. From the tonsils, and running along the ventricular aspect of the superior and inferior cerebellar peduncle, without adhering to these, the tumor extends into the ventricle and over the left side. Probing its way between the cerebellum and middle lobe, the tumor caused a central curvature towards the right of the medulla. The cerebellar hemisphere is somewhat smaller than the right. It is of a grayish-

red color, and presents some bloodvessels ramifying upon its surface. Under the scalp, upon the right side of the occipital bone, is a globular and soft tumor adhering to the skin, and lipomatus in character.

The accompanying cut, which is from a drawing made by Dr. Claribel Cone, my interne, shows very well the situation of the growth.

FIG. 1.

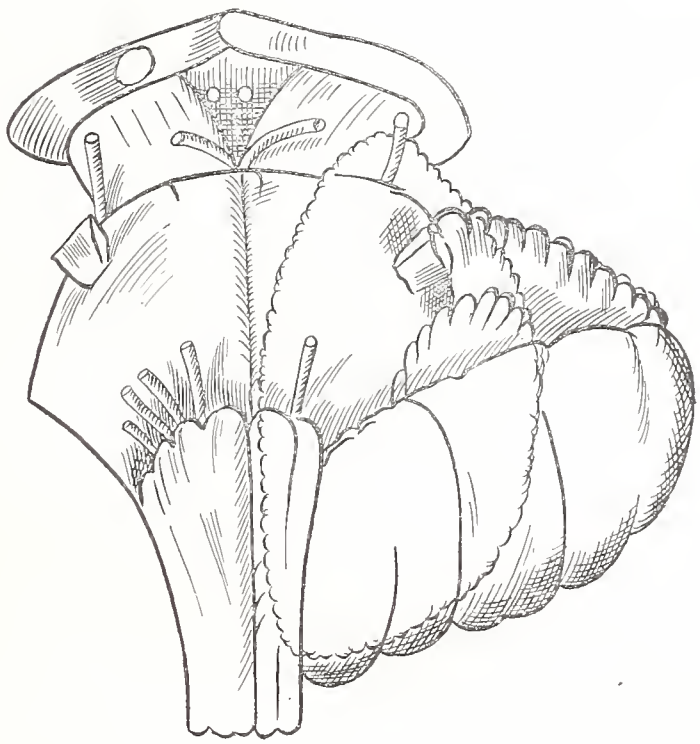


Diagram showing portion of tumor in Case I. The dotted line shows the area covered by the growth.

This case is interesting and instructive for several reasons. In the first place, on admission, the history of a sea-faring life and more or less dissipation made it seem probable that the patient was suffering from general paresis. The delusions of grandeur, and his continual boastfulness, seemed to confirm this idea. The unsteadiness of gait and hebetude, of which the patient complained, led to suspicion of cerebellar disease. The examination of the eyes showed, on his admission, no marked changes in the discs. Somewhat later, the unsteadiness of gait increased, with a tendency to pitch to the right side; and this, with the ptosis, somnolence and vomiting, comfirmed the opinion of cerebellar disease. The patient's vision was preserved until within a short time before death.

The origin of the disease in this case is difficult to explain. The violent blow which the patient declared he had received upon the occiput may have been the cause of the lesion. The lesion was very extensive, and it is only remarkable that there was not more paralysis than existed.

CASE II.—This patient was at times under the charge of my colleagues, Dr. Mills and Dr. Lloyd. The former will refer to the case elsewhere.

Biasco Madona, aged fifteen, white, born in Italy, by occupation a baker. Admitted to the eye wards of the Philadelphia Hospital, February 20, 1895, under the care of Dr. C. A. Oliver.

Family History.—Father died from injuries received in a duel. Mother still living in Italy. Three brothers and two sisters living and well as far as known. Two or three died of unknown causes.

Previous History.—Had measles and whooping-cough. When four years old was seized with severe pains in the lower extremities, followed by complete paralysis for two years. It is said that his feet were swollen and that he had fever. He recovered from this, and was very well until about one year ago, when he had a repetition of above symptoms. He speaks of having ecchymotic spots over his body and face at that time.

Onset.—About two weeks ago patient suffered from intense pain all over head. Pain was also present in the knees. At the same time his vision became dim—the failure of sight first being noticed in the left eye. The vision grew progressively worse, until patient became blind. There was vomiting at first, but no vertigo. There were no convulsions, but a short time before he became totally blind there was a short time in one day in which he lost consciousness. Before he became blind he never fell, nor was there any staggering or pitching in his gait.

Examination on Admission.—A small, well-nourished, stumpy boy; blonde, intelligent face. Temperature, pulse and respiration normal. The patient complained of nothing. The genitals are very small, and pubic hair is just making its appearance. There is no mark of any lesion on the glans penis discoverable, and the boy denies ever having had any relations with the opposite sex. In walking there is no staggering gait or pitching movements. There is, however, a little uncertainty in his gait, and this is probably due to the blindness. Tactile sensation is normal everywhere. Boulimia is a marked symptom.

Reflexes.—Knee-jerks decidedly lessened on the right side, slightly lessened on left. Dynamometer, right, 46; left, 44.

Examination of Eyes by Dr. Oliver.—"There is a small area of light-perception directly ahead, slightly larger in the left. Marked neuro-retinitis in both eyes, the disc being swollen to 5 D. in right eye, and to 4 D. in the left eye, the consecutive atrophy being more marked in the left eye. The retinal arteries are markedly diminished in size, and the corresponding veins are tortuous and full, and carry dark blood. There is marked perivasculitis in each eye—less marked in the left. Pupils sluggish in reaction, especially in right eye, which is the larger; extra-ocular muscle-action apparently good in all directions, although there is a slight divergence in the left eye, the eye being a trifle higher than its fellow."

March 11th.—Patient had attacks of vomiting which came on during the night previous. These were thought to be due to overeating. About 10 A. M. to-day he had a general convulsion; no aura preceded it. Much nausea and vomiting continued during the day, accompanied by severe headache, which was at times general, but at other times most severe below the occiput. He referred the pains to the arms.

March 21st.—He now complains of severe pain in the back of the head and neck. This is much increased by movements of the head and also of the arms. There is no rise of temperature.

The patient was transferred to the nervous wards March 23, 1895. An examination at this time was made, and the following conditions noted:

Patient lies upon his back, the head being held rigidly, apparently spastically retracted, but the neck muscles do not appear to be rigid when manipulated. The head is slightly twisted, the chin being towards the right, the occiput to the left. The patient does not seem to have much pain when perfectly still in this position, but attempts to move the head from side to side causes great pain. Careful extension seems to relieve the pain somewhat. There is some paresis of the right side of the face; there is no paralysis of the leg or arm. He has pain above the clavicle and down the arms. Coughing causes evident pain. Diagnosis made at this time was cerebellar tumor with localized meningitis in the neighborhood of the foramen magnum, with inflammation of the nerves at their points of exit.

Treatment.—Ice to the head; potassium iodide, 5 grains three times a day, to be increased; inunctions of mercurial ointment, 15 grains twice a day.

On the 26th the following note was made:

Knee-jerks are now absent, but the muscle-jerks are normal; no clonus; biceps and triceps reflexes are normal on both sides; chin-jerk normal; plantar reflex present. Patient has been vomiting considerably, but does not complain of nausea. He lies quietly most of the time, but occasionally cries out with pain, which is aggravated by the least movement. The iodide was increased to 20 grains three times a day, but as there was evidence of stomatitis the mercurial ointment was stopped.

March 28th.—Patient suffered so continuously, and with such severity from pain in the occipital region, that five Spanish leeches were applied over the seat of pain. The result was immediate and most satisfactory. The pain on motion largely disappeared after the leeching, but the vomiting still continued. Patient improved from this time, and April 12th he was out of bed and walking about the ward. He complained occasionally of slight headaches, but said there were no attacks of acute pain in the head. There was no rigidity of the neck. In walking, there was a slight tendency to stagger from side to side, but this seemed probably due to loss of vision.

May 2d.—The iodide had to be discontinued a few days ago on account of constant nausea. To-day there is a return of terrific pains in the head, just above the eyes. In the occipital region he complains of great soreness and pain, and, upon examination, this region was found to be painful to the touch, and evidences of fluctuation were present.

May 4th.—A slight incision was made into the occipital region, and a small amount of pus drained out.

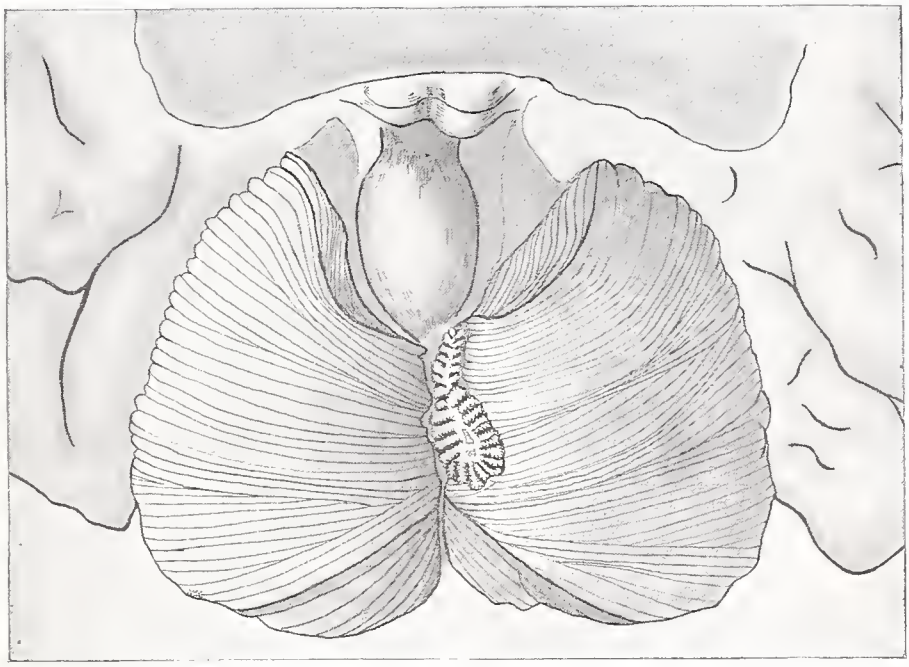
May 5th.—The patient again complained of excessive pains in the head, and five leeches were applied to the occiput, but no relief followed their application, so that morphia had to be given to control the pain.

May 11th.—Dr. de Schweinitz examined the eyes, and reported on subsiding optic neuritis and beginning atrophy. Vision is completely absent.

From this time the patient grew progressively worse, the symptoms all becoming aggravated, and he died on June 21st.

Post-mortem, by Dr. Lloyd.—Brain.—The scalp and skull show nothing abnormal. Cerebral hemispheres are normal in appearance—not unduly congested. Pia apparently healthy. On the left side of pons, and springing from it, is a soft gliomatous mass of the color and general consistence of brain matter. This neoplasm involved the exits of the fifth, sixth and seventh nerves. It extended in a lateral direction, so as to include apparently the left middle cerebral peduncle, springing from the under surface of the left cerebral hemisphere and pons, apparently in one mass. On cutting through the vermis, this mass is seen to extend upward along the floor of the fourth ventricle, and including the left anterior cerebellar peduncle. It is limited anteriorly by the posterior quadrigeminal bodies, and is confined strictly to the left side, bulging, however, well over to the right cerebellar peduncle. The mass is soft and brain-like in appearance and consistency. On laying it open from above, just back of corpora quadrigemina, it is found to be soft and gliomatous in character, with blood extravasated in its tissue. On its under surface, in the region of the fourth ventricle, it seems to have attachments to the upper surface of the medulla. It seems to have infiltrated to a limited extent the tissue of the left cerebellar hemisphere. The corpora quadrigemina and basal ganglia are unaffected.

FIG. 2.



Tumor of cerebellum.

A CASE OF DIFFUSE MYELITIS FOLLOWING SPASTIC AND CHOREIC SYMPTOMS OF THREE YEARS' DURATION, WITH AUTOPSY.

By F. X. DERCUM, M.D.

Because of the obscure origin of many chronic degenerative and inflammatory affections of the nervous system, the history of the following case, together with the pathological findings, may not be uninteresting.

M. P., female, aged nineteen, unmarried and of Irish birth, was first examined by the writer in April of 1893 at the St. Agnes Hospital, in Philadelphia. The family history was negative. Personal history as follows: Had measles, scarlatina and whooping-cough in early childhood. Menstruated at seventeen. No illness of moment in her youth. History otherwise negative.

History of Present Disease.—Two years ago the patient noticed that her legs began to get stiff so that she could not walk as steadily as formerly, and that she frequently stumbled. The condition appeared to grow worse with time. She had at no time pain, numbness, nor any difficulty with the sphincters.

Examined in April, 1893, at St. Agnes' Hospital, in Philadelphia, she presented the typical symptoms of a spastic paraplegia. Both legs seemed equally affected. Knee-jerks were decidedly plus on both sides, though there was no ankle-clonus. There were no sensory symptoms whatever, no pain, no anæsthesia. The gait was decidedly spastic. Some flattening of the left foot was noted as compared with the right. Arms and trunk appeared normal. No head and no eye symptoms. On September 20, 1893, she was admitted to the Philadelphia Hospital. Examined again the spastic condition of the legs was noted as before; knee-jerks plus on both sides, as before; ankle-clonus was now present, though slight on both sides; other symptoms negative, as before. Some time after admission slight choreic movements were occasionally noted in the arms and head. However, they rapidly became more marked and extensive. Hysterical stigmata were repeatedly sought for, but at no time found. Mentally, she was somewhat depressed, and was often tearful. With the onset of the chorea, a more or less rapid and marked deterioration of strength made its appearance. Fowler's solution, in ascending doses, made no impression upon the chorea. In October, 1893, weakness having become very decided, she was put to bed. The choreic movements were now practically continuous and very marked in extent. For a time they seemed to be favorably influenced by a mixture of bromide of potassium and antipyrin. On November 1, 1893, she distinctly lost control of both sphincters, and at this time also a slight

pustular eruption made its appearance over the coccyx and lower part of the sacrum. In a few days this was followed by an ulceration, at first superficial, but soon deep, of the the tissues over the coccyx. This rapidly spread over contiguous parts of the right buttock. It was observed that the loss of tissue in the parts invaded included at first little more than the epithelium, but soon it involved the true skin and subdermal tissues.

On November 5th her condition was again carefully studied. Both legs were now found markedly paretic, the right almost completely paralyzed; knee-jerks not as pronounced as before. Ankle-clonus present, though feeble on both sides. Tested with pin-points, it was found that tactile sensation, though present, was somewhat diminished. Pressure-sense, as far as it could be tested, appeared to be lost. The temperature-sense was well preserved. Slight choreic movements were also observed in the legs and feet; the arms were paretic and still markedly choreic; the face was somewhat flattened through relaxation of the facial muscles; now and then choreic grimaces were observed. The tongue and muscles of the neck also presented choreiform movements. The sensory phenomena in the arms and trunk corresponded to those in the legs. Pin-points were appreciated, though ordinary touch was not. Very pronounced pressure with the finger had to be made before the patient recognized the impression. An examination of the eye-grounds gave a negative result. The pupils reacted normally. Subjectively, the patient complained of black clouds before the eyes. Patient's mind was clear, though at this time the memory was slightly impaired. Examination of the heart and lungs gave a negative result, with the exception of the signs of a slight bronchitis. The pulse was soft, small and readily compressible. Its rate averaged 108. At no time was there any rise of temperature. The tongue was dry and furred in the posterior thirds, while some sordes were noticed on the teeth. The color of the patient was markedly sallow, with grayish dark markings below the eyes.

Death occurred on November 7, 1893. Autopsy, November 9, 1893. The following gross appearances were noted:

Spinal Cord.—Posterior surface much injected. Vessels of the pia excessively tortuous; inner surface of dura connected with minute and delicate adhesions with pia-arachnoid. Consistence of cord apparently normal. Transverse incision into the substance of the cord revealed decided changes in color. Here and there pinkish areas were noted, at times in the white, at times in the gray matter, and at times blending the two, not limited to motor areas.

Brain.—The dura presented no noteworthy features. Veins of the pia-arachnoid exceedingly full; small vessels injected. Consistence of brain apparently normal. Vessels of base distended and filled with dark blood. Ventricles appear to contain a larger amount of fluid than normally. Walls of ventricles moderately injected. External walls seem slightly translucent, as though the ependyma were œdematous or infiltrated. The white matter of the brain seemed more vascular than normal; the puncta vaseulosa were very prominent.

Owing to the circumstances under which the autopsy was made, the various viscera were submitted to but a superficial examination, with a negative result.

Microscopical examination of the cord at various levels reveals marked inflammatory changes. The gray matter, in all portions, and the white matter, especially in the region of the motor tracts, is excessively infiltrated with nuclei. The bloodvessels are exceedingly numerous and distended with blood. As was suggested by the gross appearances, the changes, while diffused through all portions of the

cord, are accentuated here and there, most marked, however, as a rule, in the gray matter and in the adjacent columns—more especially the regions of the pyramidal tracts. A search, however, for lesions suggesting system degeneration of the lateral columns, such as might have been suggested by the clinical history, proves negative. Where the infiltration is most dense, not only have the cells of the gray matter suffered severely, but the nerve-tubules have here and there disappeared. In other portions of the sections, more particularly the peipheral portions, numerous well-preserved nerve-tubules are seen. The changes are all of them accentuated in the dorsal and lumbar regions. The pia also reveals inflammatory infiltration. This is especially noticeable in the process which enters the anterior fissure of the cord. The portions of the cord immediately adjacent reveal here and there decided infiltration. Sections of the cortex fail to reveal marked changes, though here and there the pia appears somewhat infiltrated, and here and there the vessel walls present a similar appearance. The ependyma and the walls of the lateral ventricles present appearances also suggestive of inflammatory action, though it can hardly be claimed that they are decided.

In view of the interesting findings of Dana in chorea, the history as well as the post-mortem results of this case are exceedingly suggestive. It would appear that when the patient was first seen, the process which finally eventuated in a pronounced and diffuse myelitis had already begun, and that the spastic and choreic symptoms were due to one and the same cause. The fact that the inflammatory changes are so diffuse, and that traces of them are found even in the cerebrum, suggests that they have all been due to some general and widely acting cause. Unfortunately, we are in a position, as yet, in which we can do little more than speculate as to the agencies at work in such a case; but it is not improbable, in spite of the fact that no bacteria were observed in the sections such as were seen by Dana, that the cause in the present instance was really an infection, and that the changes observed were due to some general toxic action. It is to be much regretted that the other viscera could not be studied, though even here error might have arisen from the fact that the patient had suffered from a bedsore.

THE SPINAL CORD IN PERNICIOUS ANÆMIA.

By JAMES HENDRIE LLOYD, A.M., M.D.

The recognition of the involvement of the spinal cord in pernicious anæmia is apparently quite recent. Lichtheim, in 1887, was one of the earliest observers, if not the earliest, to write on this subject. According to Bowman,[1] he laid stress upon the special character of the changes found in the cord, as distinguishing these cases from tabes. This distinction is apparently being lost sight of by some writers, for it is becoming rather the fashion to speak of pernicious anæmia being complicated with locomotor ataxia.

It should be borne in mind that cases of pernicious anæmia with involvement of the spinal cord are neither clinically nor anatomically identical with locomotor ataxia. It is true that some of them resemble tabes more than others, but even those that resemble tabes most are not identical with it. On the other hand, some of these cases are quite distinct from tabes, and have been placed by some writers in a separate class as presenting a type somewhat similar to disseminated sclerosis.

Minnich,[2] in 1892, published a paper based upon six cases, and illustrated with diagrams of the changes in the cord. In this paper it is quite evident that these cases vary considerably in type. The symptoms in some cases resembled tabes; in some, disseminated sclerosis. Progressive weakness was marked in all cases, whereas ataxia was seen in only one-half of the number. Loss of the knee-jerks was seen in only two cases, in only one of which was there the Argyll-Robertson pupil. In only two were there subjective sensory changes, while anæsthesia, though frequently observed, was not seen in all. Loss of control of the sphincters was seen in one-half of these cases toward the end. In these six cases of Minnich's the anatomical

1 Brain, Summer Number, 1894, p. 198.
2 Zeitschrift für klin. Med., vol. xxi. 1892.

findings, as shown in the diagrams, varied as well as the clinical phenomena; they all showed a wonderful uniformity, however, in the changes found in the posterior columns. These changes were found in Goll's columns, extending across into Burdach's columns, but invariably leaving a line of normal white tissue along the edge of the posterior horn. In most cases, also, the degeneration did not extend up to the gray commissure. The root-zone almost always escaped. The variations in Minnich's cases were in the areas involved; the posterior columns were invariably involved, as just described, but in some cases the process stopped, or almost stopped there, while in others the type was rather that of a more disseminated process, involving (especially in Case V.) the direct and crossed pyramidal tracts. There was no shrinking in the posterior columns, as is seen in tabes.

Other cases published since Minnich's—notably, Bowman's and Burr's—have adhered to this peculiar type of posterior sclerosis, the root-zone and Lissauer's tract escaping. Bowman's case, however, presented also degeneration of the crossed and direct pyramidal tracts.

Bowman describes the earlier process as a swelling of the medullary sheath (to three or four times the diameter of a healthy fibre). The swollen sheath does not take the Weigert stain readily. The axis-cylinder appears normal. This description of Bowman's accords closely with what I have observed in a recent case, before I had read his paper. I have described and depicted these changes in the following pages.

The history of the case is as follows:

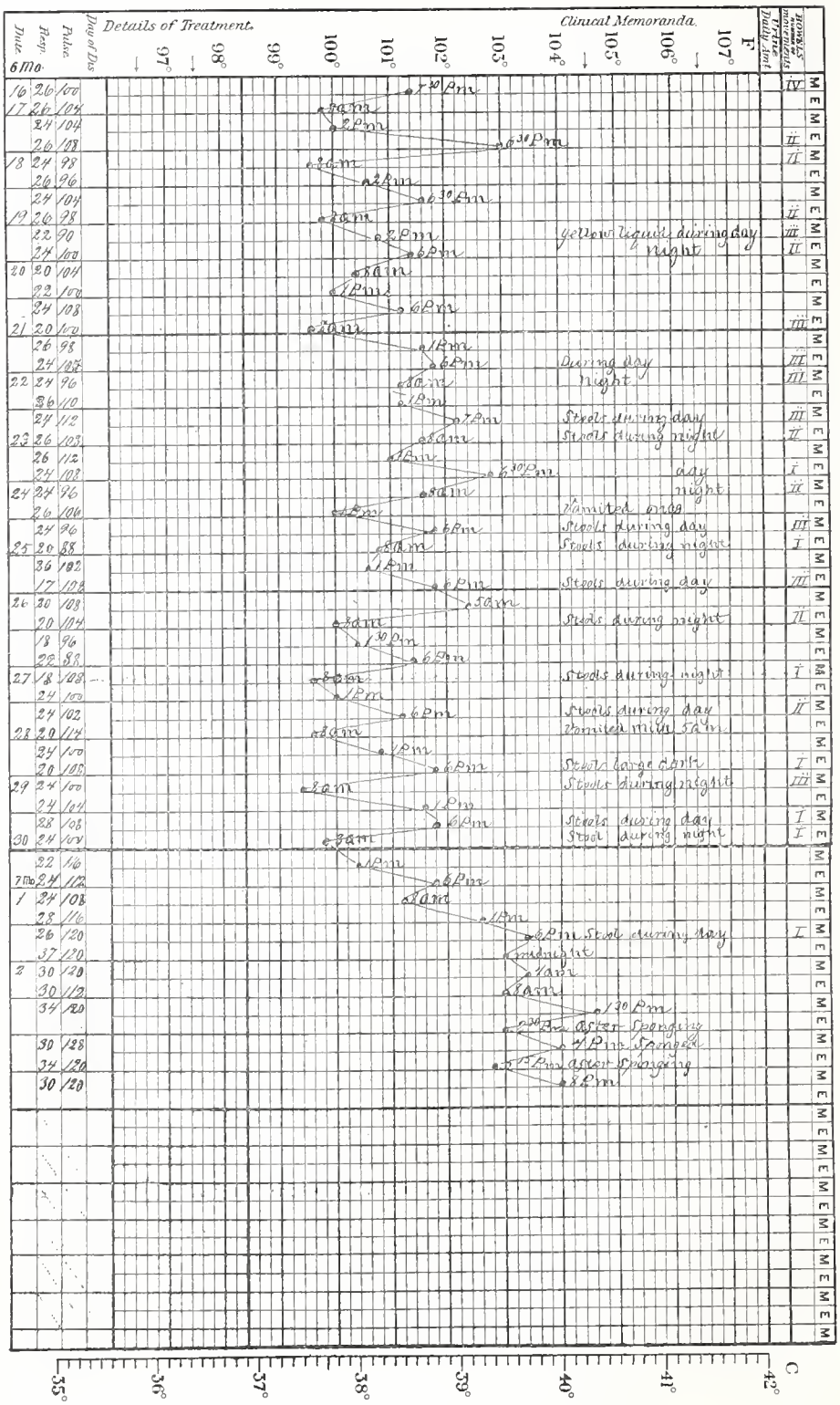
S. C., thirty-seven years, white, Ohio, machinist, single.

Family History.—Father suffered much from malaria, and died of apoplexy at sixty-four years. Mother and one sister died from a chronic diarrhœa similar to the one that the patient has at present. One sister died of some abdominal tumor complicated with diabetes mellitus.

Personal History.—The patient in boyhood suffered from malaria in his home on the Ohio river, but he has never had a recurrence since he moved to Philadelphia twenty years ago. In July, 1893, he had what he describes as a severe attack of gastro-enteritis, from which he never completely recovered, the diarrhœa persisting until his admission into the hospital. He has had typhoid fever twice. He had "inflammation of the bowels"[1] when nineteen years old. He has been a hard drinker, and gives a history of syphilis of fifteen years' standing.

He is a machinist, but he gives no history of exposure to lead of any kind. For the past two years he has been unable to work on account of increasing weakness. He has consulted many physicians and attended dispensaries without relief.

[1] Appendicitis (?) The patient had marks of wet cups over the lower abdominal walls.



Temperature Chart in a case of pernicious anæmia.

Throughout this period he has experienced cold sensations running up and down his legs, also "prickings" in the same regions, and a feeling as if he was "walking on cotton." He has had, he says, "loss of power" in his legs, but he does not give any history of ataxia in his gait; he has always moved as well in the dark as in the light. He has had girdling sensations about the abdomen. There have never been any subjective eye-symptoms, nor involvement of the rectum or bladder.

On the patient's admission, May 1, 1894, it was noted that the knee-jerks were normal. Ankle-clonus was absent. Sensation was normal. The man was very weak and could not walk straight. At this time he was already markedly anæmic, The lips were colorless, the skin a peculiar lemon-white hue, and the fat of the body fairly well preserved. After his admission it was noted that he was subject to attacks of suddenly occurring localized œdema, affecting first his legs and ankles, and then suddenly leaving them and affecting his face.

The patient complained of pain in his abdomen, centering around the umbilicus, worse after a stool.

The diarrhœa was rather irregular; at times profuse, with a large number of liquid stools in succession, followed by an interval of from six hours to several days, during which the bowels were not disturbed. He occasionally passed a little blood in his stools. He had no hemorrhoids nor history of them. He never coughed nor had any subjective pulmonary symptoms.

Examination, June 16, 1894.—The patient's skin is of a lemon-yellow color. Subcutaneous fat is fairly well preserved, and general nutrition is good. The conjunctivæ are very pale and of a pearly-yellow white, and are thrown into folds when the eyes are rolled, as the finger is pressed against them. Knee-jerks are about normal. Ankle-clonus is absent. The gait is that of a very weak person, but it is not truly ataxic. The pupils are equal; they respond to light and accommodation. Speech is normal. The tests for sensation are not satisfactory, as the man apparently cannot fix his attention. Sensation, however, is apparently not markedly impaired. The mental processes seem to be dull and confused, and to be growing worse, but are probably not disturbed more than can be explained by the patient's anæmic condition.

Chest.—Apex beat, in the fifth interspace one-half inch inside nipple line, is slightly diffused. Cardiac dulness is increased to right, extending to right border of sternum. At the apex a rough systolic murmur is heard transmitted to the axilla. In the tricuspid area a softer blowing murmur is heard over the xyphoid cartilage. No murmurs are detected in the aortic or pulmonary areas. There is marked pulsation in the suprasternal notch and in the vessels of the neck. Loud venous hums are heard on both sides over the jugulars. The pulse is rapid (100–115), rather full, soft and easily compressible.

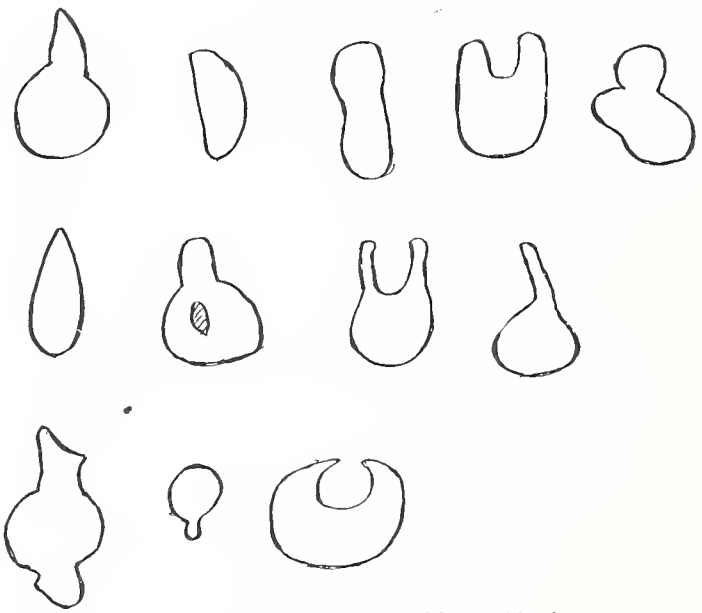
Lungs.—The sounds are rather weak, but nothing abnormal is detected. The liver is about normal in size. The spleen is somewhat, but not markedly, enlarged. At present the patient is having from four to six stools in the twenty-four hours, watery and light-yellow. Under the microscope a few pus-cells, a very few cells resembling red blood cells, and granular matter, are found in the stools. Rectal examination gives negative results.

Blood.—Red-cells = 1,280,000. Hæmoglobin, 23 per cent. Macrocytes, microcytes, and many poikylocytes, with some nucleated red-cells, are shown. (See Fig. 1.)

Urine.—Amber in color; specific gravity, 1020; acid. Chemical and microscopical examinations reveal nothing abnormal.

June 23, 1894.—The blood was examined by Dr. Daland to-day. He reported as follows: "The blood as it emerged from the puncture was pink in color. Its consistency and coagulability were lessened. The hæmatokrit showed 16 per cent. (800,000 to c.mm.) of red-cells, and slight excess of white-cells. The red-cells were of good color. Many poikylocytes with characteristic pear and flask-shapes were seen. The leukocytes seemed a trifle increased in number, perhaps 12,000 to 15,000. The smaller-sized leukocytes predominated in number; many were about the same size as the red-cells, and rarely was a large white-cell visible. The white-cells seemed filled with coarsely granular material. Some were irregular in shape, and occasionally one was seen to throw out pseudopods." Count of white-cells (3040 to c.mm.).

FIG. 1.



Forms of poikylocytes noted in the blood.

June 24.—An examination of the eyes was made to-day by Dr. de Schweinitz, who found the vessels of both retinas diminished in size and the retinas the seats of many hemorrhages.

July 1.—Red cells = 648,000 to c.mm.

July 2.—Spots of extravasated blood have appeared at the base of the sacrum. The patient is much weaker. Cerebral anæmia is very marked. Carphalogia has been present at intervals for the past week.

July 3.—Death at 6 A. M.

Post-mortem Notes. July 4, 1894, thirty hours after death.—Body of fairly nourished man. Skin of lemon-yellow color. Subcutaneous fat is well preserved and of an orange color. It measures three-quarters of an inch in thickness in the abdominal wall. Tissues seem practically bloodless, and muscles are highly colored. No œdema of tissues is noted. The marrow of the lamina of the vertebræ is soft, but not fluid; it is dark-red in color. The abdominal cavity does not contain fluid.

The *appendix* is adherent at the tip, as if it might have been the seat of an old inflammation, but does not show signs of any recent disturbance.

The *liver* seems slightly diminished in size, extending barely to the costal margin, is yellow on section, and appears fatty.

The *spleen* measures 6 x 4 x 3 inches. Capsule is somewhat thickened and wrinkled. There are four notches on the edge of the organ, giving it a serrated appearance. On section the tissue is dark and soft, and the pulp is rather easily squeezed out.

The *kidneys*, on section, appear to be fatty. The cortex is about the usual thickness.

The *pancreas* is normal.

FIG. 2.

The spinal cord in pernicious anæmia. Dorsal region.

Intestines.—No enlargement of mesenteric glands is notable. No marks of ulcers; no tubercles nor tumor.

DESCRIPTION OF SECTIONS.—*Stains.*—(1) Carminate of ammonia; (2) Hæmatoxylon; (3) Methyl-green; (4) Weigert; (5) Biondi-Heidenhein.[1]

POSTERIOR COLUMNS.—*Dorsal Region.*—The degeneration does not occupy the whole of the posterior columns. In the dorsal region the area of degeneration is about midway between the posterior median septum on either side and the posterior horn of the same side; hence it involves part of each postero-median and postero-external column. (Columns of Goll and Burdach.) This area does not extend quite up to the posterior gray commissure, the normal white matter with the cut ends

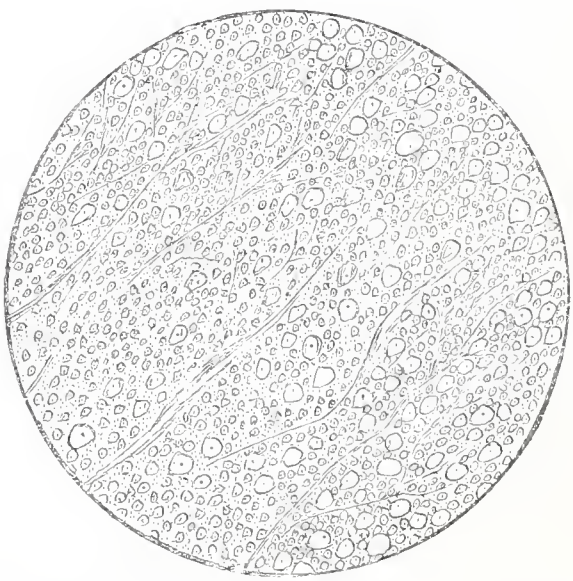
[1] Cervical region not removed with cord.

of the nerve-fibres being conspicuous around the anterior edge of the degenerated area. Around the post-median septum the degeneration is not so dense as at a slight distance away from it on either side. At the point of deepest degeneration no normal tissue can be traced; *i. e.*, no nerve-fibres are visible. Hence the area of deepest degeneration may be described as spread around the division line between the columns of Goll and Burdach on either side and not extending quite up to the posterior commissure in front nor quite to the periphery of the cord behind. The posterior root-zone is not seriously involved in this cord—a fact which accords with the clinical findings of no ataxia and of preserved knee-jerks. Still these trunks do not escape entirely, the most median ones being involved toward the periphery, before they emerge from the cord.

Lumbar Region.—Almost, if not quite, normal in the posterior columns.

Lateral Columns.—The lateral columns have been studied very carefully. In the dorsal region, as shown especially in the section stained with methyl-green, it

Fig. 3.



The spinal cord in pernicious anæmia. From the lateral columns, showing swollen nerve-fibres.

is evident that some change has occurred in them. This is shown (1) by an increase in the neuroglia, and (2) by great distention of some of the nerve-tubules. In some of these distended tubules the axis-cylinder can still be seen; in others, not. This distention of the nerve-tubules, it seems to me, is of great significance. It probably shows an early stage of degeneration, in which some irritant is acting upon, or even infiltrating into, the tissues. In the first stage the tubule swells; then the axis-cylinder breaks and finally shrivels; and, finally, the neurolgia, which has been proliferating all the while, begins to contract and blot or squeeze out the true nerve-tissue. In this cord the early stage is probably seen, in which the most conspicuous feature is the swelling of the nerve-tubules. In the lumbar region, where, of course, the lateral columns are not so large as in the dorsal cord, these columns are not nearly so markedly involved. These swollen tubules may also be seen in some of the degenerated tissue in the posterior columns.

Deiter's cells show with unusual clearness in the posterior columns in some regions. They can be picked out especially well with the carmine stains.

The membranes are not distinctly involved in this cord.

The anterior horns, with their multipolar-cells, are normal in both dorsal and lumbar region. The cells are conspicuously free from disease in all the sections.

NERVE-TRUNKS.—These cannot be said to be markedly affected so far as the shape and contour and the amount of interstitial tissue is concerned. With the Weigert stain, however, the fibres do not stain evenly, and this may possibly be an indication of commencing change in them.

Concluding Remarks.—The anatomical changes, just depicted, explain why these cases of pernicious anæmia do not present the identical symptoms of locomotor ataxia. As in Minnich's and in Bowman's cases, already referred to, the posterior root-zones are but partially involved. It cannot be said, however, that they escape entirely in my case. It is possible that the degree of their involvement varies slightly in different cases, and this variation may explain the absence or presence of such tabetic symptoms as the loss of the knee-jerks, the ataxia and the subjective sensory symptoms. In some cases, in which the disease-process is more disseminated, and the lateral tracts involved, the patients present the clinical picture of ataxic paraplegia. It is probable that this would have been the case with my patient had he lived longer, as the lateral tracts were becoming distinctly involved, and must soon have become sclerosed.

It seems almost useless in a purely clinical and anatomical study to speculate about the essential cause of pernicious anæmia and the relation of this cause to the changes in the spinal cord. Other writers, who have essayed to do this, have not succeeded in throwing any light upon this important subject. It is a purely pathological question, and cannot be solved apparently by ordinary bedside methods or by post-mortem histological studies. It may be permissible here, however, to call attention to a few points and to a few analogies, which may assist the future investigator.

First, it is to be noted that this patient's range of temperature is strongly indicative, according to received opinions, of some irritant or toxic substance circulating in the blood. It was continuously high for weeks before death ; for at least two weeks before the end it did not come down to the normal once. It rose steadily toward the end, registering once almost as high as 105°. When it is considered that the autopsy revealed nothing in any organ or tissue that could account for such continuous high range of temperature, the conclusion seems

almost forced that it could be explained only by an abnormal blood state produced by some toxine.

Second, the character of the tissue-change in the spinal cord is strongly suggestive of the action of a poison. The change begins apparently in the parenchyma; thus, as we have seen, the myeline sheath becomes enormously distended, and this apparently before the neuroglia begins to proliferate markedly. It may be too much to assume positively that this parenchymatous change is always the result of the action of a toxic substance, yet it is certainly in harmony with what we know already of the action of some such substances. In this connection, it will be interesting to note in the future whether the spinal cord is ever involved in a similar way in the ordinary secondary anæmias.

Finally, the persistent diarrhœa, sometimes with hemorrhage, may have some special significance in this case. Especial care was observed at the autopsy in examining the gastro-intestinal tract, but nothing was observed that could, in any way, account for the symptom.

Acknowledgments are due to Dr. J. Dutton Steele, resident physician, for clinical work done in this case.

CASES OF APHASIA, ILLUSTRATING ESPECIALLY DISORDERS OF PANTOMIME.

By CHARLES K. MILLS, M.D.

In Volume II. of the Philadelphia Hospital Reports, the writer published a short paper upon "Disorders of Pantomime Occurring Among Aphasics," giving notes of two cases. The following cases, mostly studied in the hospital, may be regarded as a further contribution to this subject.

Case I.—*Brachiocrural Monoplegia—Almost Complete Motor Aphasia—Marked Preservation of Pantomime.*

The first patient represented a common, and yet I do not think the most frequent, type of combined aphasia and right-sided paralysis, and showed retention to a remarkable degree of intelligent and expressive pantomime.

M. N., a woman, seventy years old, had been paralyzed and aphasic for fifteen years. Her right arm was helpless, strongly flexed at more than a right angle at the elbow, and the hand contractured into a bunch. The right lower extremity was almost equally paralyzed and presented marked flexure at the knee. The face showed no paralysis, and if any had existed its traces were scarcely detectable. Both sides of the forehead corrugated equally; both eyes could be firmly shut, and the mouth could be drawn to the right as well as to the left; the tongue was protruded easily and without any deflection or peculiarity of movement. The knee-jerk and tendon-jerks generally were increased on the paralyzed side. As far as could be determined, sensation, cutaneous and muscular, was normal.

The study made of her speech disturbances can be briefly summarized. She understood sounds, letters and words, evidently comprehending all that was said to her. When asked the name of "cat," she called it a "pussy." She could name also a few objects without suggestion, as "pin," "pen," "book" and "key;" but her powers in this direction were very limited, and she could not name the vast majority of objects which were familiar to her, as "cup," "nurse," etc. She, however, recognized promptly what objects were and their uses, and instantly detected the mistake of a false name given to anything before her. She pointed

out or picked up objects when requested. She was a woman of limited education, but was able to pick out nearly all the letters of the alphabet, failing to recognize a few of them, as "B" and "C," even on repeated tests. Letters in red she was entirely unable to pronounce, probably not recognizing them through some color-blindness. Her spontaneous vocabulary was exceedingly limited, consisting of a few words and a frequent recurring utterance, "come-on-to-nong." She mis-pronounced some of the words she could say. She could repeat words and brief phrases after another person, but not sentences of any length. Compared with her powers of volitional speech, however, her ability to repeat from dictation was excellent. Reading aloud, writing, either voluntarily or from dictation, and copying, seemed impossible to her, although these deficiencies may have been largely due to her want of education.

In a paper on aphasia I have recorded at some length this woman's pantomimic powers as follows: "She not only understood all that was said to her, but within the limits of her original capacity, education and experience, could, so far as her unparalyzed members would permit, express her meaning clearly and distinctly by the most significant pantomime. With the instruments which nature had left unimpaired, she could promptly indicate what she wished to convey, and yet she was tremendously crippled as far as ordinary speech was concerned, and had as a most common method of vocal reply a routine recurring utterance, 'come-on-to-nong.' Her pantomime had high propositional value. In studying her pantomimic power, for instance, I asked her age, and, with her unparalyzed hand, she opened and shut it fourteen times, the movement becoming slower and a little more emphatic as she approached the end. She told, in this, that she was seventy years old, and when I asked her if she meant seventy years old she nodded her head (yes) in a most emphatic manner. I asked her how long she had been sick, and with her hand she promptly told me fifteen years. I said to her, you mean twenty; she shook her head (no), and again shut and opened her hand three times to indicate fifteen."

As would be expected from the close topographical relations of the speech and the orolingual and other centres for the face, aphasic and pantomimic disturbances will be always more pronounced in hemiplegic cases in which facial paresis remains as a marked feature. In the present case, the tongue, lips and face showed no paralysis. In my studies of hemiplegics, I am in the habit of making a critical examination of the face and all parts of the body for the particular varieties of motor loss. Hemiplegics, whether aphasics or not, can be

arranged into classes, according to certain peculiarities in the local distribution of the degree of paralysis. Many of them exhibit a paresis of the face which can only be detected by the closest investigation. Even when the eyelids on the paralyzed side can be apparently fully closed, examination will show quite a difference in the furrows which are produced by the act of closure.

No auditory affection and neither hemianopsia nor word-blindness were present in this patient, although she seemed to have a form of color and letter-blindness, the meaning of which was not fully determined.

One of the cases cited by Bateman[1] bears a striking resemblance, with regard to the preservation of pantomime, to this patient. It is the case of a man, aged sixty-three, who was engaged to be married and was suddenly seized with right hemiplegia and aphasia, and wished to make his will in favor of the lady he intended to marry. The will was written by one of his medical attendants. The testator's mark was made and the will was attested by witnesses. He made signs for writing materials; his wishes were interpreted by means of signs and then written down on a card. He held up his hand, extended his five fingers, and was asked if he meant a "thousand;" he bowed assent. He then closed his hand and opened it in the same way, implying ten; this operation was repeated until it amounted to thirty, and then he dropped his arm down. Testator was then asked whether he wished Miss R. to have £30,000, and he nodded his head. In order that there might be no mistake about his wishes as to details, he was asked whether Miss R. was to have this sum absolutely; he signified dissent, but on being asked if it was hers for life, and afterward to revert to his family, he bowed his head. Unfortunately, the testator's mark was made in the middle of the card instead of at the bottom or foot, and so did not satisfy the provisions of the statutes relating to wills, and the testament was refused probate.

Many instances have been recorded in which cases of decided aphasia, with little or no paralysis, retained almost perfect pantomime. Boinet, for example, who is cited by Kussmaul, relates the case of a man with a small abscess in the region of Broca's convolution, who, after trephining, remained speechless, but could make himself under-

[1] Bateman, On Aphasia, etc., London, 1890. Quoted by the writer in the Review of Insanity and Nervous Disease.

stood by gestures and pantomime, playing with his comrades, copying from dictation, and writing out his own thoughts.

Our case shows that among hemiplegic aphasics we may have recovery of pantomime, performed by the unparalyzed limbs, just as we may, in other cases, have recovery of articulate language, either shortly after an apoplectic seizure or early or late during the protracted history of a hemiplegia, and that this pantomime may be recovered far beyond ordinary speech.

The positive symptomatology of this case was permanent paralysis of the leg and arm and of speech, with the preservation of facial power and pantomime, which could be exhibited by expression of countenance, by movements of the head and body, and by the intelligent use of the unparalyzed limbs. The lesion was probably not cortical, because of the character and completeness of the brachio-crural monoplegia and its association with profound aphasia, in spite of the facts that the centres for the limbs and speech are widely separated. Doubtless, it was largely one of the anterior portion of the internal capsule, and possibly the ganglia, but it embraced more. The difficulty is to show how such a lesion would impair speech while allowing the face and tongue to escape. In all probability, the fibres from the face-centres to the bulbar nuclei escaped.

In numerous instances hemiplegic patients who at first suffer from marked paralysis of the leg, arm and face, and complete or nearly complete aphasia, recover largely or almost entirely control over the face, and ability to speak as well as to use pantomime, while loss of power in the leg and arm remains. I could give from my note-book numerous examples of this form of hemiplegia, but they are familiar to all neurologists. In patients of this class, the lesion is usually situated in the posterior half of the internal capsule, involving often the lenticular body, the external capsule and other parts escaping, except that they suffer early from the effects of pressure. Our patient differs only from this large class of cases in the pronounced permanent aphasia. The auditory and visual centres and the concept areas are evidently unaffected, but either the motor cortex for speech or its subcortex, and the callosal commissures for speech are apparently destroyed. The right hemisphere has not taken on the speech functions of the left, although it has fully assumed the powers of intelligent pantomime.

CASE II.—*Hemiplegia and Convulsions—Word-Blindness—Verbal Amnesia—Motor Aphasia—Sensorimotor Disturbances of Pantomime.*

M. S., thirty-five years old, a married woman, about four years ago, two weeks before her expected confinement, was suddenly attacked with spasms which lasted off and on for nearly twenty-four hours. The child was delivered dead with instruments. For three days she was unconscious, and on coming to herself she had fever and numerous hallucinations and delusions. Nine days after the first attack of spasms, she was found to be speechless and paralyzed on the right side. She remained in bed about three months, during which time she regained some power in the paralyzed limbs and the ability to speak a few words. She continued to improve slowly in speech and limb power, although the total improvement was not great. The first year after she was paralyzed she had ten or twelve spasms, but she had no others from that time until one year ago, when she had a series of convulsions lasting altogether three hours, and afterward she was found to be considerably worse, both as to speech and the loss of power in her limbs.

Examination showed well-marked paralysis of the right upper and lower extremities, with a less degree yet decided loss of power on the right side of the face. She was unable to use her right hand and arm for practical purposes. Hemianæsthesia was not present—touch, pain, and temperature senses were everywhere preserved. Examination by Dr. Edward Jackson showed an irregular form of hemianopsia.

She evidently comprehended all that was said to her, and was not, therefore, word-deaf. She had had a common school education, but her husband stated that since her attacks she had never been able to read, and did not seem to know one letter from another. Testing her carefully, it was found that she could not recognize either letters or words, although she seemed to make strenuous efforts to do this, ending usually with a disturbed look and a shake of the head. Sometimes she would say "can't tell." She was not mind-blind, or if so, only partially, as she seemed to recognize persons and objects, but sometimes with difficulty. Of course, she could not read either silently or aloud. The effort to have her write her name was made, but she could not do this with either hand. Besides word-blindness, she had marked verbal amnesia, and volitional speech was almost lost; she had great difficulty in naming objects which she apparently recognized; she could, however, sometimes do this and could sometimes, when strongly urged, answer questions of a simple character, the reply to which required only a word or two. She could repeat a few words and short sentences from dictation, although she could not recognize by sight a single letter of the alpabet; when asked to do so she repeated every letter correctly and in order, and without mistakes

in pronounciation or enunciation. Such words as she used were not misplaced, but she did not put the words together into complete sentences when she practiced the little voluntary speech of which she was capable. In brief, her disorder, so far as speech was concerned, seemed to be an admixture of word-blindness, verbal amnesia and motor aphasia.

She was tested as to pantomime in various ways—as to her ability by this means to express voluntarily her thoughts and wishes, to do what she was told to do (pantomime corresponding to dictation), to do what others did before her (pantomime corresponding to copying). She pointed, beckoned and motioned away, but awkwardly. On telling her to close her eyes she did so promptly; telling her to lift her left hand she tried to raise her right or paralyzed hand, and, finding she could not do so, said "can't tell." Asked whether she was married, she said "yes," with the proper forward nod of assent, but immediately after said "no," shaking her head properly for dissent. Asked if she had a headache, she said "yes," with an assenting nod. Asked if she had any houses, she nodded "yes," and assented when she was asked if she had but one house; and she responded similarly to various other tests. She could assent and dissent by pantomime understandingly; she did not, however, always do this correctly, but usually corrected herself when she was wrong. Often she used the trunk and head instead of the limbs. Her pantomime usually correct, had in it an element of slowness, uncertainty and awkwardness. She could imitate correctly most things that were done before her.

An effort was made to test her testamentary capacity. She was asked to whom she would leave her money if she had any, and after two or three efforts she finally succeeded in saying, "Al," which was ascertained to be an abbreviated name for her husband. On asking if she meant her husband, she said "yes," and made an affirmative movement with her head and trunk, a favorite mode of pantomime with her. I wrote a few words and told her it was a statement leaving all to her husband, and asked her to sign her name or mark to it, which she was unable to do, but seemed willing. She was exceedingly awkward in her efforts to use the pencil in her left hand, and did not seem to know how to take hold of it for the purpose of writing.

A reference of her symptoms to the probable site of the lesion seemed to indicate that the auditory tracts and centre and the com-

missure between the auditory centre and Broca's convolution had escaped, but that the lesion was of a character and in a situation to destroy either the centre for word-images or the arc between it and the concept centres, and also that between the concept centres and the motor speech region, as well as the internuncial fibres for face, arm and leg. Her lesion was probably subcortical and capsular, the interhemispheric commissures largely escaping.

Trousseau found that aphasics also sometimes imitate gestures, but cannot make them at request unless they have been previously made before them. This is not ataxia, but *amnesic amimia*.

"The patient, Paquet, who, besides his name, could pronounce almost only the word 'cousisi,' imitated the movements of clarionet playing with exactitude, and comprehended also what they meant. When requested a few minutes later to make the same movements of clarionet playing, he reflected, but was generally incapable of accomplishing this simple pantomime."

CASE III.—*Right Hemiplegia—Marked Paralysis of the Face—Almost Total Abolition of Pantomime—Almost Total Sensorimotor Aphasia—Marked Obstinacy—Energetic Emotional Gesticulation.*

This patient, E. C., was an old inmate of the hospital, her age unknown, but probably about sixty years. The right arm was paralyzed and contractured and drawn over the chest. The right leg was contractured at the knee at more than a right angle and adducted in a dorsal position. Her face drooped slightly on the right side; she could not apparently thrust her tongue beyond her teeth, and had a tendency to retract it in the mouth. The lines and contours of the right face were not well marked as on the left. Cutaneous anæsthesia was not present, but no fine investigations of sensation could be made.

This woman's vocabulary was confined almost entirely to the word "no." In the ward she commonly goes by the name of "no-no," as another patient does by that of "la-la." She seemed to use this for almost everything—for assent, dissent, to express pleasure, anger, dislike, to attract attention, etc. It is both a recurring utterance and an occasional utterance; at times it is emotional, and again it has a propositional value. The "no" is used for both "yes" and "no," and may easily mislead. She has three other expressions used very rarely, and which she will sometimes repeat when told; these are "nonsense," "now" and "no good."

Her "no" is often accompanied with an energetic gesture, an up

and down chopping movement of the unparalyzed arm, a movement which, like her "no," is always the same. It is often exceedingly difficult to determine how much she understands, and still more to decide whether her gestures and her utterances have the value of true speech and of pantomime. At times when she means to say "yes," she accompanies the "no" with an energetic movement of the arm, and lifts the head and has a cross facial expression, which gives every appearance of strong dissent. "No" and the arm gesture are undoubtedly used to express anger, passion, pleasure, etc. She never, however, points to anything which she wishes, or beckons or motions away, although she uses the unparalyzed hand for such matters as brushing away flies, feeding herself, etc. When obstinate or angry she will sometimes cover her mouth with the unparalyzed hand, apparently with the purpose of expressing disgust or dissent. She often laughs as if she were amused, particularly after she has been told to do things.

CASE IV.—*Marked Hemiplegia of Gradual Development—Motor Aphasia and Anarthria—Slight Degree of Loss of Pantomime.*

This case is of special interest for the reason that an autopsy was obtained:

M. D., fifty-five years old, had several times been an inmate of the hospital, but was last admitted in June, 1891. She was first carefully examined by me four months after her admission. When first admitted she had some paresis of the right side of the body and a slight disturbance of speech. The loss of power and disturbance of speech had gradually increased. Her mental condition was one of slight apathy and indifference. Owing to her mental condition and her speech defects, it was somewhat difficult to get satisfactory results in examining her for sensation, which was impaired somewhat on the right half of her body. She complained greatly of pain in her paralyzed arm and leg, particularly when they were handled. I might say in passing that this pain and hyperæsthesia, which is comparatively common in the paralyzed limbs of hemiplegics, is often due to a neuritis set up either by the injuries to which the helpless limbs are liable, or from pullings and twistings of the nerve-trunks or plexuses, the result of muscular relaxation and the weight of the limbs. The paralysis of the right upper and lower extremities was complete, and was of the flaccid type, without contractures, although the fingers showed a slight crooking at the first and second phalangeal articulations, probably not a true contracture. This absence of contracture, in view of the site and character of the lesion afterward discovered, is of interest.

Paralysis of the right side of the face was very marked, much more so than in most hemiplegics and monoplegics. The mouth was decidedly twisted to the left. She could not protrude her tongue, and when she opened her mouth it lay almost

motionless with the point turned down just behind the teeth. She had some use of the organ, however, as will be seen, both for speaking and other purposes.

Knee-jerk was increased on the paralyzed side. Occasionally, even at this period, she had incontinence of fæces and urine. This woman lived for three months after examination, and the following notes were taken later: Her paralysis, speech disorder and mental apathy steadily increased. Examinations were made on several occasions, but the results of these were not essentially different to those given here. Toward the last nothing satisfactory could be determined. She became much emaciated, and sank into a state much like that of a patient suffering from melancholia atoneta. She was thoroughly tried with anti-syphilitic aud other remedies.

She was examined before a class of students as to her disturbances of speech and pantomime. She was not sound or word-deaf, and was not mind-blind. She had never learned to read or write. Volitional speech was much impaired. This showed itself particularly in her attempt to answer questions; she was often unable to do this, even about the simplest matters, but she could talk spontaneously to announce her wants and wishes with considerable facility. Her difficulty was not one of recalling and rehearsing words and sentences, but in arranging and uttering them. Sometimes she used emotional language freely, indulging in oaths and angry expressions. She could repeat remarkably well anything dictated to her. Her answers to questions were sometimes correct and sometimes not. While she sometimes answered incorrectly, on testing her with statements which were plainly false she could respond properly. Her voice was low and her mode of speech slow, sometimes stumbling, and sometimes halting between words and syllables. As she grew worse and worse, her ability to make herself understood became greater and greater, her voice lower and weaker. Until a very late period she seemed to know what was said to her, and to know what she wished to say herself. She would stumble over difficult words, but could pronounce many simple words with ease. Pantomime was unimpaired except in so far as it was affected by her general apathy and weakness.

Autopsy.—Nothing abnormal was found in the skull or dura. The pia-arachnoid was decidedly opaque and milky-looking over both hemispheres in the postero-frontal and antero-parietal regions for a distance of a couple of inches from the median line, the appearance being more marked on the right thau on the left, and it was most decided over the upper extremities of the central convolutions and superior parietal convolutions. This membrane stripped off with great ease from the cortex. In the Sylvian fossæ, also external to the origiu of the middle cerebral arteries, the pia-arachnoid was opaque.

On opening the left lateral ventricle the head of the caudate body was apparently normal. The thalamus presented a bulging and distorted appearance, as if displaced inward and forward, presenting a knobbed or nodulated appearance at its anterior internal extremity. Behind and within the thalamus, just where the caudate body bends down toward the middle horn, was an area one and five-eighths inches long and one and one-quarter inches in breadth, of a dark-reddish and yellowish-brown appearance. It looked as if it covered an old hemorrhagic cyst. At one place it was slightly depressed.

Transverse sections were carefully made, beginning at the head of the striate bodies. For a distance of about one and one-quarter inches the caudate and ventricular bodies, the capsules, claustrum, etc., were perfectly normal, but at this distance from the head of the ganglia a cut revealed by a dark grumous streak the beginning of a tumor. The limits of this mass were afterward traced by a series of transverse sections. In its anterior portion it invaded the inferior and median portion of the ventricular body. Half an inch further back the growth invaded and infiltrated the entire lenticular body and external capsule, pushing aside the internal capsule and thalamus. It was found here to consist of a collection of whitish and yellowish-brown nodules, infiltrated with blood. Another section showed that the mass reached as far as the posterior limits of the thalamus, invading here not only the external capsule and lenticular body, but the internal capsule and, to some extent, the thalamus. The whole of this posterior half or third of the mass was broken down and infiltrated with recent hemorrhage. The growth did not at any place reach the outside of the brain. It was probably syphilitic, but has not yet been examined microscopically.

Marked pleuritic adhesions were found on the right side; caseous nodules were found in both apices, but no cavities. The heart was small and flabby; the kidneys were small, scarred, and granular; the spleen and liver were normal; the ovaries and the uterus were atrophied. The other organs were not examined.

CASE V.—*Double Hemiplegia from Successive Lesions on the Left and Right Side of the Brain—Absolute Abolition of Speech and Pantomime.*

The most striking illustrations of the destruction of speech, gesture and pantomime—every means of communication from the patient to others—is seen in some cases in which successive or simultaneous lesions causing hemiplegias and aphasias have occurred on both sides of the brain. A patient is stricken with right hemiplegia and aphasia nearly or quite complete; he recovers the power of speech and pantomime in a measure, and it may be to a less degree muscular power in the paralyzed face and extremities; he is now attacked with a second apoplectic stroke which paralyzes the other side of the body, and we have, in fact, an instance of so-called double hemiplegia, and with it a total loss of all ability to communicate by speech or muscular movements. During a recent term of service in the Hospital, I had a case in my wards.

This man, M. P., aged forty-six, a laborer, was admitted to the hospital May 21, 1891. He was then paralyzed in the left leg and arm, with loss also of sensation more marked in the arm than in the leg. The arm showed contracture, although slight movement had returned to it. Shortly after admission he was found to have intercapsular fracture, which probably occurred at the time of the hemiplegic attack, but was first disguised by the loss of power and the fact that the man was completely bedridden.

Three months after admission, while the patient was lying quietly in bed, he was suddenly taken with convulsive movements involving both sides of the body. His head deviated toward the left; his countenance was anxious; the left pupil became very slightly larger than the right. Aphasia was complete. The tongue could not be protruded. Temperature was at first, in the right axilla, 98.8° F.; pulse, 110; respiration, 124; temperature in the left axilla, 99.4° F. He remained in this condition about twenty minutes, when the convulsive movements stopped. He seemed to understand everything that was said to him, although he could not speak. He was looked over the next day. His right arm was then completely paralyzed like the left, and his right leg was also at least partially paralyzed. Some paresis was present in the right side of the face, and his mental condition was curious. He looked at a person as if with recognition, but could not be induced to make any response to anything said to him. He lay apparently conscious of his surroundings, but absolutely speechless and expressionless, and devoid of all pantomime. When he did not see the person speaking it was difficult to say whether he heard what was said to him.

Case VI.—*Motor Aphasia and Amnesia—Word-Deafness at first—No Paralysis—Convulsions—Nearly Complete Amimia—Mental Dulness and Confusion.*

C. C., aged sixty-five, white, born in Ireland, a bricklayer; during July, 1892, had two attacks, probably spasms, but not described. On October 10th, while sitting on a sofa, had a fit, in which he was unconscious. The hands were firmly clenched and there were clonic movements. On recovery he had loss of speech, memory, and the power of understanding what was said to him. He had had rheumatic pains for a long time. He used alcohol and tobacco to excess. He was admitted to the hospital thirty hours after his attack. He was then able to be moved about, and showed a tendency to wander aimlessly.

His left eye showed the result of an old keratitis; arcus senilis moderately marked; radials fibrous; temporals scarcely visible and sclerosed.

It was difficult to make out fine differences, if they existed, owing to his mental condition, but there was no paralysis of the face or extremities to be determined, even of slight degree. The case was one of aphasia without motor or sensory paralysis. Sensation to touch, pain, and temperature preserved. Knee-jerk and muscle-jerk present—about usual.

The following notes were made a few days after admission:

The patient is evidently suffering from a high degree of mixed aphasia and paramimia. Usually he seems to be word-deaf. In two or three instances he

answers simple questions with a proper negation or affirmation, or by a movement of the head; but generally he does not answer at all, or with a meaningless jabber of half coherent words, as if he took some other meaning. Possibly when he answers correctly it is accidental or from seeing the mouth of the questioner, or by putting together circumstances

He recognized words in large letters, as the *Philadelphia Inquirer* on the newspaper; pronounced "Inquirer," sometimes perfectly and sometimes imperfectly. He has difficulty in recognizing words in small type, but he is blind in one eye and hypermetropic in the other. He is certainly not completely object-blind, and undoubtedly he quickly recognizes objects by touch.

In conversing, he shows a peculiar tendency to become absorbed in some single idea; he will continue to repeat in slightly varied form the same sentence, string of words, or even jumble of syllables; these are usually suggested by persistent questions, and when once he becomes absorbed it is almost impossible to divert him, but if ordered to be quiet he usually obeys. After a period of rest he becomes involved in some new suggestion. Certain objects and certain persons are more easily suggested than others, and will often be indicated by the patient himself. The most prominent of these are his son or two sons, and particularly his own malady, which he locates in the tongue and believes to be due to a mechanical restraint of that organ. When first admitted, he answered all questions and orders with "Tungis tie" (tongue-tied). Nothing indicating appreciation of his surroundings could be elicited. Even amimia was complete.

Three days later he is much more intelligent, often answering surprisingly well complicated questions. More frequently he makes a jumble of words, using several words that would form part of an appropriate sentence; sometimes he only attempts to respond to a small percentage of the questions as if they were no more than a mere stimulant to psychical activity, repeating for answer a subject that was previously engrossing him. He answered his name readily, but could not tell his age; when asked if he were 200 years old, he said "yes." On being told to hold up a pencil, he did so at once. On being told to hold out both arms he would not do so, but held them out after they had been placed in position. When told to touch his head he did not do it; the right hand being placed on his head and removed, he was then told to do the same with the left, and did so a trifle slowly. Asked to give the color of some white cloth he became confused, spoke several meaningless syllables, and finally said red.

Just before dinner time he came to the office and explained, partly by signs and partly by incoherent speech, that he had nothing to eat. Shown a clock, the hands at 11.53, he, after considerable effort to direct his attention to it, suddenly appeared to become more intelligent, and said 1.10; and then, without appearing to appreciate that he contradicted himself, he said 10.30. This seemed to satisfy him, and he went away, after being assured he should have his dinner.

When asked where his son was, he answered promptly, "Where is my son? I don't know where he is." When asked from the side, so that he could not see the interlocutor's mouth, if his tongue troubled him, he answered at once, "Why, there is nothing the matter with my tongue." Shown a plate of bread without butter on it, he seemed to want something to put on the bread; he did not seem to want to eat it, or else did not understand what it was for; he repeated several times, "What is this for?"

Two weeks later the following notes were made:

When one of the physicians had examined him in the ward, he looked after him and remarked, "I wonder where I have seen that man before?" His partial word-deafness has steadily and rapidly improved. Apparently, he silently reads and understands as far as his imperfect sight will permit. Attempting to read aloud, the effort is gibberish. He still has great difficulty in putting words into sentences of his own volition. From dictation he repeated one simple sentence nearly correctly: "I am an old man"—"I am a sin man." Following this sentence with others, he showed a continuous tendency to return to the original sentence, occasionally mingling words with the old sentence from the new. His spontaneous language was gibberish or jargon, and also his attempts to read aloud or speak from dictation, although he showed considerable variation in his speech.

He shows very little tendency to use affirmative or negative pantomime, but does occasionally, and better than at first. Attempting to have him express himself affirmatively or negatively, he seemed to be confused by the idea, and it was impossible to make him use pantomime by telling him to do so, or through imitation. He was offered a due bill and a form of bequest to sign; it seemed to puzzle him, but he did not sign either.

November 26, 1892—four weeks later—the patient was out on liberty, and when he returned he was in a stupid, semi-conscious, unresponsive condition, with a few scratches and bruise-marks on his face and hands; he gave the impression that he had been on a spree. He remained in this way over night, and the following day had to be restrained in his bed because of his desire to get up in his semi-clad state. He was kept in bed for a few days, when it was found that restraint was having a baneful influence. His speech and power to express himself were much worse than when he went out. He was very restless and fidgetty, and acted just like a child, doing many things he ought not to do, and leaving undone many which he should. When he was accused of drinking he denied it with all the vehemence he could muster, and got down on his knees as if to take an oath he had not. He became very devoutly inclined, getting down on his knees frequently and commencing a prayer, which, after a few appropriately expressed words, became meaningless jabber. On December 28th it was noted that he persisted in not wearing shoes and stockings or coat, throwing down suspenders with the evident intention of taking off his trousers if opportunity offered. He had to be cautiously watched. Soon after this, he was removed to the insane department where he died, but, unfortunately, no autopsy could be obtained.

A SERIES OF REPORTS OF CASES FROM THE NEUROLOGICAL DEPARTMENT.

SERVICE OF DR. CHARLES K. MILLS.

FIRST SERIES.—I. HYSTERICAL HEMIANÆSTHESIA; SUCCESSFUL TREATMENT BY METALLOTHERAPY.—II. SYPHILITIC DISEASE OF CERVICAL VERTEBRÆ WITH SPINAL HEMORRHAGE; INTERESTING LOCALIZING PHENOMENA.—III. OLD LENTICULAR AND PONTILE CYSTS; RECENT PONTILE HEMORRHAGES.

REPORTED BY CHARLES A. VANDERVOORT, M.D., RESIDENT PHYSICIAN.

CASE I.—*Hysterical Hemianæsthesia; Successful Treatment by Metallotherapy.*

D. G., aged nineteen, Russian, a tailoress, single.

Onset.—Two months ago, while working in a jacket factory, she began to suffer from headache and gradual loss of power in left arm and leg. She continued to work until ten days ago, growing gradually weaker on the left side.

Family History.—Good. She gives an indefinite history of one sister having attacks of pain in the head and vertigo.

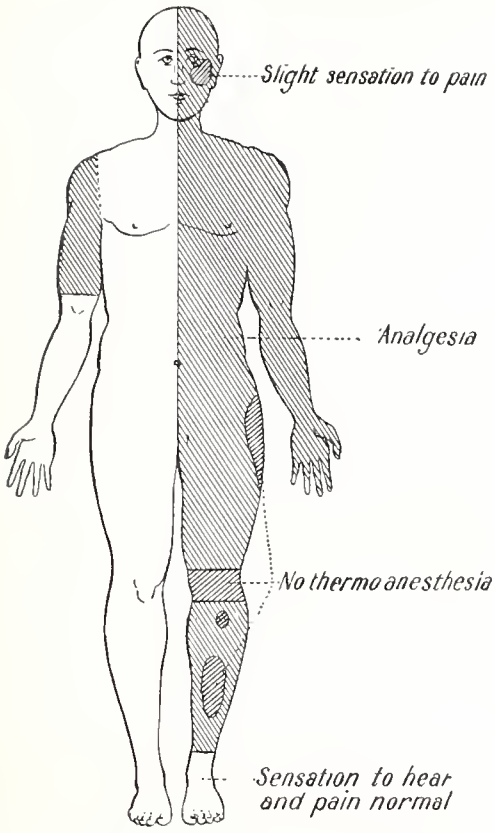
Personal.—Patient is a good-looking Jewess, well nourished, black eyes and hair, and cheeks constantly flushed. The upper and lower incisors are distinctly notched.

Almost every expiration is attended by a moan. She complains continually of pain in the head, worse on the right side. Expression, variable; at times sad, but she can be easily provoked to laughter. The extremities of the left side are distinctly paretic. She is unable to stand without support. The paresis affects some of the facial muscles also, particularly about the forehead and eyes. Tongue clean and protruded in the median line.

With the exception of the foot and a small area over the malar bone, the entire left side of the body is analgesic and anæsthetic. This extends to the exact median line of the body, including the left half of the tongue, nose and chin, and the left labia majora.

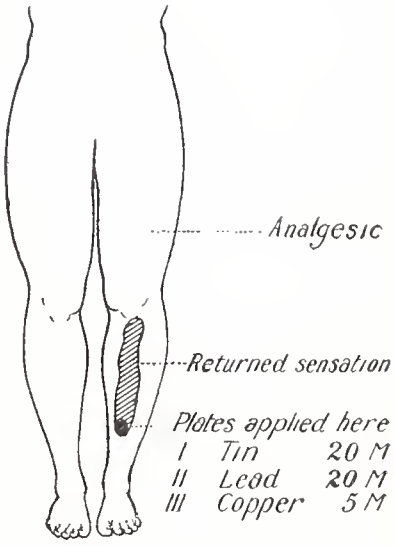
In the foot response to irritation is slow but present, as over the left cheek in a small area. Perception to heat and cold is lost on the same side, except in two areas around the hip, knee, and a small area over the tibialis anticus. The entire

FIG. 1.



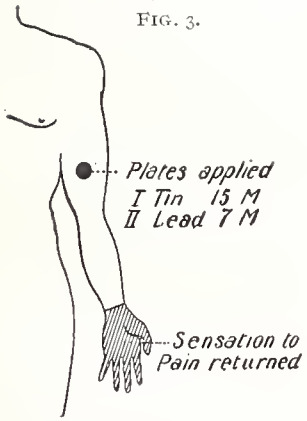
Hysterical hemianæsthesia; anæsthetic areas before the metals were applied; areas of analgesia all shaded, and thermo-anæsthesia present except the small areas of deeper shade; the shaded area of the upper right arm became anæsthetic two days after first examination of the patient.

FIG. 2.



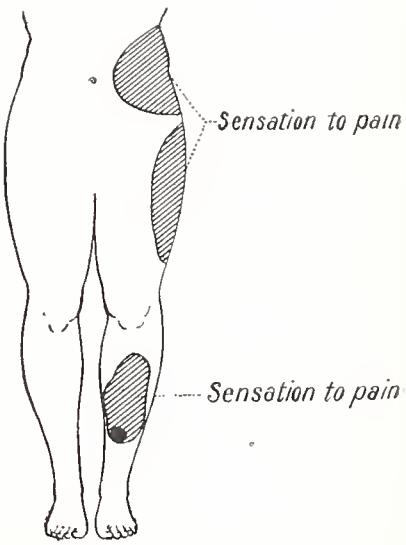
Results of first application to the anæsthetic leg of metal plates, September 20, 1894.

FIG. 3.



Results of application of plates to the anæsthetic arm, September 21, 1894.

FIG. 4.



Results of continued applications, September 22, 1894.

RESULTS OF METALLOTHERAPY IN A CASE OF HEMIANÆSTHESIA.

right side of the body is hyperæsthetic both on deep and superficial irritation. This is marked over the ovary, beneath the breast, and along the spine. The muscles of the left side respond fairly well to the faradic current. The response over the right side is exaggerated. Pupils regular, equal, and respond well to light and accommodation.

About two hours after admission she complained of intense pain in the right upper arm and shoulder, and declared it felt just like the left side of the body early in the disease. By examination this area was found anæsthetic, but not paretic.

Menstruation was normal; there was no sphincter paralysis. Examination of the eye-grounds by Dr. Gould showed nothing but anæmia of the fundus.

This being evidently a case of hysterical anæsthesia, etc., the method of treatment known as metallotherapy, used largely by some French physicians, was determined upon.

Accordingly, on the afternoon of the 20th treatment was begun as follows: Plates of iron, brass, copper, zinc and lead, two inches in diameter, were secured. A given portion of the body was examined and found to be completely anæsthetic. The plates were then applied in succession to the same part, each remaining on a certain length of time, while the patient was blindfolded.

Experiment I.—September 20th, 1.40 P. M. Anterior surface of the leg used, the plates applied midway between the ankle and knee.

Tin applied at 1.40 P. M., removed at 2 P. M.

Lead applied at 2 P. M., removed at 2.20 P. M.

Copper applied at 2.20 P. M., removed at 2.25 P. M.

About five minutes after the copper was applied the patient pulled up her leg, and said the metal burned her. On removing the plate there was both tactile and painful sensation beneath it, and extending as far as the knee as a strip two to three inches wide. There were no transfer phenomena. Twenty-four hours later this had extended over all the anterior surface and sides leaving a strip over the posterior surface unaffected.

September 21, 1894.—Experiment tried over thigh to-day was performed as above, but no changes in sensation noted.

Shortly afterward the plates were applied as before to the upper arm. Tin plate remained fifteen minutes, followed by the lead one for five minutes, when she complained of pain and tingling in her hand, which was found normal. No transfer phenomena, but beneath the plates the parts were normal.

Under further applications the areas rapidly extended, the paresis disappearing, and she was discharged practically well, September 29, 1894.

CASE II.—*Syphilitic Disease of Cervical Vertebræ with Spinal Hemorrhage; Interesting Localizing Phenomena.*

J. D., a colored man, aged thirty-seven, a native of Philadelphia, a laborer by occupation, and married.

Onset.—During the afternoon of September 4th he had carried about twelve tons of coal from the street to the cellar, had his evening meal, and sat down upon a doorstep to rest. He shortly experienced severe pain in the back, soon followed by general weakness. He became partially unconscious, fell upon the ground, and lay there in a stuporous condition for some time before he was able to call for help. At that time he noticed he was powerless in all the extremities except the left arm.

Family History.—Nothing of importance or of interest.

Personal History.—Has always been a hard worker and a heavy consumer of alcohol and tobacco. Several years ago he had an attack of acute rheumatism, but he denies all venereal disease.

Present Condition.—The patient was brought to the hospital in an ambulance about 5.30 P. M., September 5, 1894. He was a very large, muscular man, of fair intelligence. He lay flat upon his back, legs rotated far outward, right arm prone and extended along the side. These three members, when lifted, fell heavily and helplessly. The left arm was held in a peculiar position. It was lying on its posterior surface, slightly abducted, and flexed at the elbow and wrist in such a manner that the hand hung in the air over about the junction of the two upper bones of the sternum. His abdomen was slightly distended. His respirations were rapid, labored, and entirely diaphragmatic. Speech thick and difficult, the latter due probably to respiratory embarrassment. Pulse good and regular. Temperature normal. Physical examination disclosed the following:

The right arm, the muscles of the chest and abdomen, and the legs were completely paralyzed. Motion was impaired in the right arm, but certain movements could be quite easily executed. When the arm was placed in contact with the side, he could abduct it sufficiently to place the elbow five inches from the side. When the arm was only partially extended he could again flex it, but was unable to do so when fully extended. He could not pronate the hand and wrist, but could slightly supinate it. He was able to protrude or retract his arm by the shoulder muscles as a whole. All other attempts at movement of this arm were useless. The head and neck could be moved with ease, and his tongue was protruded in the median line.

There was some tenderness over the back of the neck to the mid-scapular region. No sensations of pain. From the second interspace and downward the entire body was analgesic, except over the deltoids and the biceps of the left side. Over the legs, responses to temperature changes were very confusing and incorrect, and he called the hot tube cold much more frequently than it should have been. This was noticed over the entire body, but not so markedly as over the legs. Tactile sensation was fairly well preserved, the patient usually detecting the finger or pencil when applied to the skin—not so readily, however, over the legs as over the trunk and arms.

Epigastric reflex slightly present; left bicipital present; all others were absent except conjunctival.

Bowels involuntary, urine retained.

The patient was placed upon a water bed and kept perfectly quiet. The surface of the body was cold and moist.

Seven hours after admission he suddenly complained of violent pain in the back. Immediately after, his temperature shot up to 103° and he soon became delirious. Respirations became shallow and were of a modified Cheyne-Stokes character. Tympanites became excessive.

September 6th.—This morning he seemed better; mental condition improved as well as general condition, except the tympanites. About noon, however, he began to vomit, the material being first yellow, then brownish in color. During the night of the 6th and the day of the 7th the vomiting continued to grow worse, assuming a slight fecal odor. The tympanites grew to an enormous extent, and the patient's bowels could not be moved. He gradually sank, and died at 5.30 P. M., forty-eight hours after his admission.

Autopsy twenty-four hours afterward.—Permission was obtained to remove the brain and cord only. A clot about three inches in circumference was found beneath the scalp, its centre corresponding to the lambda.

The brain was normal. On removing the muscles and vertebral laminæ, the spinal canal was found full of dark fluid blood as high as the junction of the fourth and fifth cervical vertebræ. A small subdural ecchymosis was found at the anterior surface of the cord about opposite the origin of the seventh cervical nerve. The disc between the fourth and fifth cervical vertebræ and the bodies of the vertebræ adjoining were soft and spongy, lacked the dural lining of the canal, and were the seat of old disease, probably syphilitic. The diseased area was nearly the size of a nickel, and presented an appearance altogether unlike that of any other portion of the canal.

This case is an interesting one from several points of view:

1. The onset after a very hard day's work.

2. The position of the partially paralyzed left arm, said by Thorburn to be characteristic of this condition.

3. The exacerbation of the symptoms so soon after his admission, probably due to the outpouring of a fresh quantity of blood from the diseased area of bone.

4. The escape of portions of the nerve-roots from which came part of the supply to the left arm, due in all probability to a lower level of the hemorrhage in the spinal canal on the left side.

Thorburn, in his "Contributions to the Surgery of the Spinal Cord," quotes a case to which, except as to causation, this almost exactly corresponds. The case referred to was due to fracture of the fifth and sixth cervical vertebræ, and it was shown at the post-mortem that the pressure exerted on the cord was sufficiently high on the left side to involve the fifth nerve-root, which escaped entirely on the right side.

In this case this could not be so definitely demonstrated post-

mortem, but the symptoms presented were sufficiently characteristic to indicate that the pressure on the cord and nerve-roots was higher on the right side than on the left, involving the nerve-roots entirely on the right side, and but partially if at all on the left.

CASE III.—*Old Lenticular and Pontile Cysts; Recent Pontile Hemorrhages.*

A. S., aged sixty-nine, married, a bartender, a native of Pennsylvania.

Onset.—Always enjoyed good health until two years ago, when he had a fall, sustaining scalp wounds of the front and back of the head. These healed rapidly, but the patient's friends state that he has not acted altogether right ever since. In January, 1893, he had a "stroke," after which the tongue and the right side of the body were paralyzed. He regained partial use of the arm and leg, as well as of the tongue, but has had quite marked impediment in speech since that time.

He has always drank heavily and steadily, rarely going on a "spree." No history of venereal disease could be obtained. He is the father of nine children, six of whom are living.

The patient was admitted to the nervous wards September 22, 1894, in a stuporous condition from which he could be aroused, recognizing his surroundings for a short time but soon again getting stupid. When aroused he would try to talk, but only a few words could be understood.

He was fairly well nourished, face puffy, legs slightly œdematous, right side paretic. Plantar, cremasteric, and epigastric reflexes present. Plantar slightly plus; no ankle-clonus. No areas of anæsthesia or analgesia could be found.

His heart and lungs were normal, excepting an accentuated second sound over aortic cartilage. About six ounces of urine were secured by catheterization, which on examination was found to be about one-eighth albumin by bulk, and containing dark annular and hyaline casts. Pupils reacted to light and distance and were equal and normal in size.

The symptoms pointing more toward uræmia than any other trouble, led us to treat the case as such. This was done by purging, hot-packs and diuretics.

The day following admission his eyelids were puffy, œdema of the legs greater and respiration shallow and stertorous.

Small amount of urine removed by catheter was found about one-fourth albumin by bulk. Treatment was continued.

The following day the tongue continually fell back far in the throat, impeding respiration so much that it had to be held forward by a stick. The patient's condition grew worse, and death occurred forty-eight hours after admission.

The *post-mortem* was made twenty-four hours after death, and the particular points of interest were as follows:

1. Three pontile hemorrhages.
2. Minute cysts of the pons.
3. Cyst of the left lenticular body.

In left lenticular body, on a level with the middle of the thalamus, a cyst was found about the size of a pea. It was close to the internal capsule, toward the upper surface of the lenticular body, and filled with clear fluid.

In the left portion of the pons, between the median line and outer border, three

minute cysts were found extending from the anterior portion backward, growing somewhat larger and disappearing in the posterior third. Three distinct hemorrhages were found, two being situated on the left and one on the right side of the median line. The first was disclosed by transverse section, about 3 mm. above the junction of the ventral aspect of the pons and oblongata. It was on the left side, irregularly oblong in shape, the larger diameter being placed transversely and measuring about 3 mm. It was just beneath the centre of the left half of the pons, and had disappeared entirely 3 mm. above the first section. Another very small hemorrhage (2 mm.) was found on the right side, deeply situated, at about the junction of the lower and middle thirds. About 3 mm. from junction of the left crus with the pons still another was found, about $1\frac{1}{2}$ mm. in diameter, situated deeply in its substance.

SECOND SERIES.—I. Oculomotor Paralysis; Exudate Surrounding Right Internal Carotid Artery and Adherent to Optic and Third Nerves; Occlusion of Right Internal Carotid and Posterior Communicating Arteries; Recent Clot in Left Internal Carotid.—II. Paralysis of Left Internal Rectus of More Than Sixty Years' Standing.—III. Abducens Paralysis; Marked Emotional Disturbances, and Various Mental and Paretic Phenomena—Small Focus of Embolic Softening in the Dorsal Pons; Prefrontal Tumor; Cyst of Right Lenticula and Internal Capsule; Cyst of Left Lenticula.—IV. Numerous Apoplectiform Attacks Causing Paralysis, Areas of Local Anæsthesia, Speech Disturances and Paralysis of the Iris on One Side.

Reported by H. R. GAYLORD, M.D., Resident Physician.

Case I.—*Oculomotor Paralysis; Exudate Surrounding Right Internal Carotid Artery and Adherent to Optic and Third Nerves; Occlusion of Right Internal Carotid and Posterior Communicating Arteries; Recent Clot in Left Internal Carotid.*

L. S., aged twenty-nine years, born in Philadelphia, a seamstress.

Father died of unknown cause; mother died, when the patient was young, of a tumor.

The patient had measles and whooping-cough and scarlet fever when a child. She has been a hard drinker for the past three years. She had both ovaries removed in the hospital two years ago.

In June, 1894, she had sore throat and alopecia, but does not remember having had a rash or chancre previous to that time.

In July, 1894, she began having persistent frontal headache, which has continued up to the present time and has been somewhat relieved at intervals by repeated courses of potassium iodide. She has had at various times shooting pains of a severe type.

From July her eyesight began to fail, and on awakening in the morning of September 4, 1894, she noticed for the first time that her eye turned upward and outward. On attempting to rise she was unable to stand, and discovered that her

left leg was useless, sensation, however, returning by degrees. She regained some use of the leg, and excepting occasional attacks in which it would suddenly give way, she was able to get around on it; finally she was obliged to take to bed, complaining of feeling generally depressed.

December, 1894.—Patient has a rather stupid expression of countenance and wanders about aimlessly and speaks but little; constantly complains of headache, which she says is inside her head. She has spells in which she becomes irritable, followed by marked mental hebetude, and then takes to bed and is not easily roused and will not answer questions. Her gait appears shuffling, rather more suggestive of indolence than weakness. Her station is good. Knee-jerk plus and slight ankle-clouus.

Examination of Eyes by Dr. Oliver.—Right eye ptosis complete. Eye directed out 45 (about).

Can fix with right eye, but cannot bring eye to centre; movement of globe lost, except out, and slight wheel motion up and out.

Left pupil 2.5 mm., responds to light.

Right eye pupil 4 mm., immobile.

Pupil oval, long axis 90.

Disc oval, 7 by 8, axis 90.

Scleral ring outside of disc externally.

Disc gray throughout its substauce, most marked to the temple side.

Left eye, pupil oval, long axis 95. Disc oval, 7 + 8 long, axis 90, same tint as disc of right eye, gray iu the deeper layers, especially outside.

February 28, 1895.—Patient weut to bed in a spastic condition similar to previous attacks, but more marked. The left side of the face is flushed and swollen; she will not speak except in monosyllables. Temperature during the night remained about $100\frac{4}{5}°$ F. Both legs are cold. During the night she had an attack resembling convulsions, followed by general muscular twitchings. Third nerve paralysis is deepened; she will not answer questions, although she appears to understand.

March 1, 1895.—Meutal dulness increased, gradually deepening into coma; will not take medicine; lies in the position in which she is placed.

March 2, 1895.—Respirations rapid, shallow and blowing; right arm stiff, left relaxed; both legs relaxed. Knee-jerk exaggerated on left side as compared to the right. Left eye open and fixed, pupil contracted. Does not take food or medicine. Coma marked. On pricking the arms with a piu the extremities are drawn up.

12.30 P. M.—Nearly pulseless. Temperature $104\frac{3}{10}°$ F. No reaction to pin-prick in the right leg, left leg reacts.

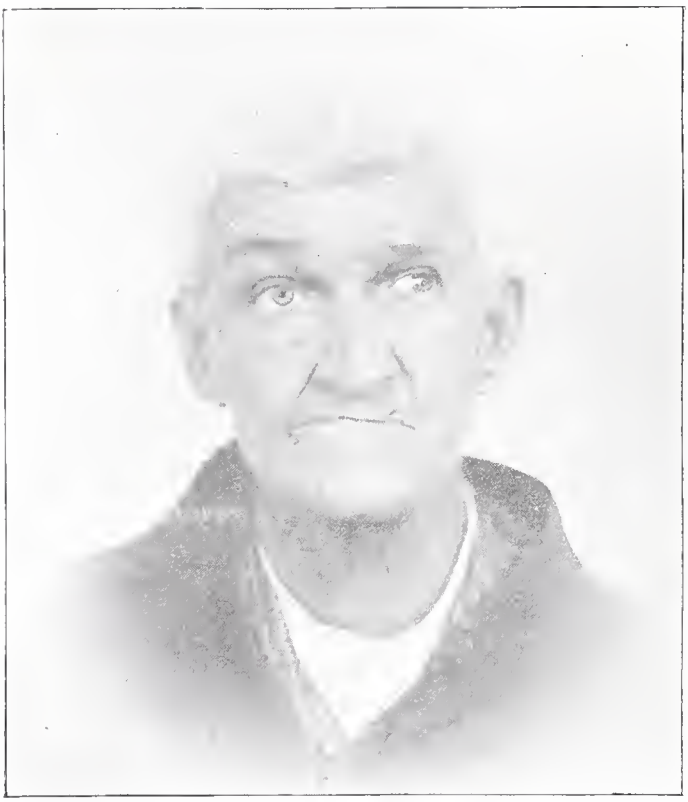
2 P. M.—Cheyne-Stokes respiration for about thirty minutes; is pulseless at wrist. Right arm spastic, stiff twitchings noticed iu both arms; left arm straight and stiff. Left eye partly open, turned upward and outward. Extremities cold and cyauosed up to wrist and ankles. No reaction to pin-prick anywhere over the body. Breathing stertorous, a few fine râles in the lungs auteriorly.

Died at 8.30 P. M., with no change in conditiou.

Autopsy.—Left kidney 120 grammes; capsule slowly removable. Thickness of cortex slightly reduced, aud is pale. Malpighian bodies plainly seen. Centre of kidney contaius some fat. Right kidney 105 grammes, otherwise same as left.

Brain.—Calvarium slightly increased in thickness. Diploë distinct. Meningeal

FIG. 1.



Photograph showing left external strabismus without ptosis. Paralysis of left internal rectus of more than sixty years' standing. (Case II.)

vessels all show distinctly on the surface. Dura normal, not adherent. The pia is free from inflammation and thickened throughout. The vessels over the hemispheres are distended. The right internal carotid is free until within two or three millimetres of the penetration of the dura, and the plug in the artery is flesh-colored and well organized.

The right posterior communicant is a mere filament, anterior cerebral very small. The left anterior cerebral is very large. The left posterior communicating is very large, much more so than the post cerebral. To the right side of the chiasm and the beginning of the optic nerve there is a dense yellow mass surrounding the internal carotid and adherent to the optic nerve and third nerve; the mass lies in the beginning of the fissure of Sylvius, which it fairly obliterates. The left internal carotid also contains a clot in the region from which the posterior communicating and anterior and middle cerebral form. This clot also extends into the middle cerebral artery about one-half its length.

The cerebellum, pons and oblongata on section show nothing abnormal.

On making a long section of right hemisphere the entire convexity showed no lesion. The head of the caudate nucleus, the anterior extremity of the striate body and lenticular nucleus softened. The material in the softened cavity is liquid and about the color of pus. The section of the right hemisphere does not show any gross lesions.

Case II.—*Paralysis of Left Internal Rectus of More Than Sixty Years' Standing.*

C. L., laborer, aged sixty-seven, single.

Parents died of old age. Sister died of phthisis. Two brothers died from wounds received in the Civil War.

He has had rheumatism since boyhood, and at various times has been confined with inflammatory attacks. Had scarlet fever when a child, and following the attack his left eye was turned upward and outward.

Examination.—Hands show deformity of rheumatoid arthritis.

Vision.—O. D., $\frac{2}{15}$. O. S., count fingers at 30 cm.

O. S.—Eyesight has been poor ever since patient was one year old, following scarlatina, and eye turned outward since. Scar on cornea triangular in shape.

Iris tissue somewhat degenerate and greenish in tint; iris freely mobile to light. Pupil, 2 mm. O. D., iris freely mobile to light. When O. D. is made to look to the right O. S. turns right and in (paresis of left internus).

When O. D. is made to look up O. S. turns up and out.

When O. D. is made to look down O. S. turns slightly down and out. In conjugate deviation to right O. S. internus fails to respond fully. When O. D. is made to fix upon fingers brought along median line to nose, O. S. turns slightly up and out. (Fig. 1.)

Examination with the ophthalmoscope shows high myopia in left eye. Vision brought to one-ninth by proper correction.

Eye-ground shows characteristic change of high myopia. Right eye the same.

Left field shows marked contraction, especially to the nasal side.

It is impossible to get right field, as patient refuses to submit to further examination.

Pupils the same size.
Irides freely mobile to light.
Paresis of left internus.

CASE III.—*Abducens Paralysis; Marked Emotional Disturbances, and Various Mental and Paretic Phenomena—Small Focus of Embolic Softening in the Dorsal Pons; Prefrontal Tumor; Cyst of Right Lenticula and Internal Capsule; Cyst of Left Lenticula.*

L. B., aged fifty-three years, Philadelphia, druggist. May, 1894.

For the last three years the mother, aged eighty-nine, has had spells, in which she falls down and becomes unconscious. The rest of the family history is good.

Patient had malaria twenty-eight years ago; about twenty-six years ago had a sore on penis, but no secondary eruption. Fifteen years after the sore, for which he had taken mercury, he had small ulcers below the knees, which were called syphilitic by his doctor. He was a great drinker for a long time and also a great smoker.

History of Present Disease.—About eight months ago a white film came over the left eye and it became turned upward and inward. He had had no strabismus previously. He lost to a degree the power of vision in the right eye. He had at no time much pain in the eye, and he recovered the sight in part under treatment at the Polyclinic Hospital. He saw double for a while.

He soon after fell in the street several times from weakness and dizziness and had to walk with a cane. His left hand and leg became gradually weaker than the right.

He has been free from headache and pains. While talking he frequently bursts into tears. He says he cannot help it, and does not do so because he feels especially bad. His walk is like that of a left hemiplegic.

Dynamometer.—R., 32; L., 18. Knee-jerks +, especially the left leg. No ankle-clonus or anæsthesia.

February 22, 1895.—Patient has entire loss of station but is able to walk slowly by pushing a chair in front of him.

There is not atrophy of muscles of left side, but loss of power in left arm and leg, although he has some use of both. R., 45; L., 22. There are no areas of anæsthesia, both legs are rather hyperæsthetic and all reflexes increased, having marked patellar and muscle-jerks, and plantar reflexes and ankle-clonus present.

His thermal sense is good. He complains of no pain but feels week. He continues to have emotional outbursts, which he says have no relation to his feelings.

Examination of the eyes as follows: The vision of the right eye reduced to one-half; left to almost one-third. It is impossible to measure the fields on account of the mental condition of the patient. Both nerves are slightly off color, especially in their temporal halves. The retinal arteries are reduced in size. Accommodation-power good in each eye. There is paresis of right externus. (Fig. 2.)

Autopsy.—The calvarium is not thickened. The dura was so adherent that it was necessary to remove brain in calvarium. There is a small area of erosion of the inner surface of the calvarium to left side of median line and one at the anterior extremity of the calvarium.

On removing the brain from the skull there is found a tumor involving the

FIG. 2.

Case of pontile lesion, showing paralysis of the external rectus and expression of the face. (Case III.)

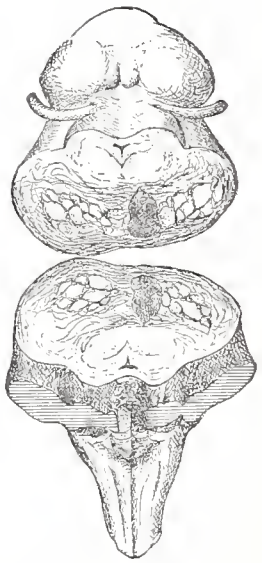
dura and one from the anterior extremity of the first and second frontal gyri of right side. This tumor does not extend into the brain substance more than half an inch. The mass is moderately firm; color, yellow. It appears to be of uniform density throughout.

The pia of the hemisphere can be removed readily on making a transverse section of the right hemisphere; there is a cyst from softening involving the head of the internal capsule and the outer extremity of the lenticular nucleus. It is one and one-half inches long.

After section of left hemisphere a cyst is seen from softening involving the lenticular nucleus. It begins with a transverse section which cuts off the anterior extremity of the temporo-sphenoidal lobe. This cyst is about half an inch in length and breadth, depth less than one-half inch. No other lesion found in hemisphere. Cerebellum shows no lesion.

The pons shows an area of softening on its left side near the median line in the post half. The above lesion was revealed by a cut 15 mm. caudad of the junction of the pons and crus. It was close to the median line, entirely in the dorsal half of the pons; its width from median line laterally is 5 mm. and in a cephalo-caudad direction about 7 mm.

FIG. 3.



Drawing showing the position and apparent size of the pontile lesion in the fresh specimen from Case III. in the text.

FIG. 4.

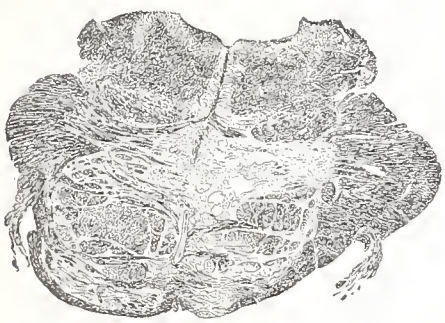


FIG. 5.

In Figs. 4 and 5 are shown the microscopical appearances of the lesion in Fig. 7. The degeneration is seen to extend across the median line and over a large area.

CASE IV.—*Numerous Apoplectiform Attacks Causing Paralysis, Areas of Local Anæsthesia, Speech Disturbances and Paralysis of the Iris on One Side.*

H. R., age forty-three years, married.

Healthy parents. Family history negative.

Had syphilis twenty-two years ago.

Disease began in 1887, in July. While at work felt a trembling sensation in the fingers of the left hand, followed by numbness and loss of power. This was

followed in a few minutes by a general weakness and loss of power in which he fell to the ground, but was conscious of his surroundings although he had lost the power of speech.

From this condition he gradually improved, and in two weeks had regained some power on left side and could speak somewhat, using only a few words. In six weeks was able to get around on crutches.

In four months he had regained speech, and was entirely well except that the right arm was weak, and in walking the movements of the right leg were uncertain.

November, 1887, he had a second attack while hunting. He was climbing a fence when he was suddenly taken with an attack of loss of power and unconsciousness. Several hours afterward he had lost power on the right side entirely and speech was again impaired.

He recovered from the second attack, except that when speaking in a hurry he would use words not related to what he was saying and was still weak on right side.

He was admitted to the hospital January, 1888, in about the same condition as above described.

On May 30, 1889, he found that he was unable to stand without support, and on examination anæsthesia of the right leg was first noticed. He was confined to bed several months following this attack.

In July, 1891, he had a similar attack.

September, 1892, he had a second attack of paraplegia, and at this time he first noticed that there was trouble with his eyes. He noticed that one pupil (right) was larger than the other (Fig. 6), and his general condition has remained the same, until now it is as follows:

Has some power in his legs, can walk with cane.

Gait.—Swings left foot outward and drags the toe as he lifts it from the ground. Right leg he carries forward stiffly and straight, tilting the pelvis to the left to allow it to swing forward. Has loss of power in right arm, which hangs helpless at his side. There are marked contractures of flexors of right hand and some atrophy of flexors; deltoid markedly atrophied.

There is no anæsthesia except on the back portion of the right calf for a space not over two inches by eight inches; he is not able to judge of distance between points, and cannot tell how the ground feels.

Heat and cold sensation are normal.

Skin reflexes on legs are increased.

Patella reflexes are increased on both sides; on right side tapping on tibia or above and below patella will throw the leg into clonic spasm, lasting as long as three minutes if not checked.

There is left ankle-clonus.

Irritation of the skin on the right leg will throw it into spasms.

Station lost, cannot walk at all. Some atrophy of the right thigh.

The examination of eyes shows that fields of vision, the vision and accommodation are normal.

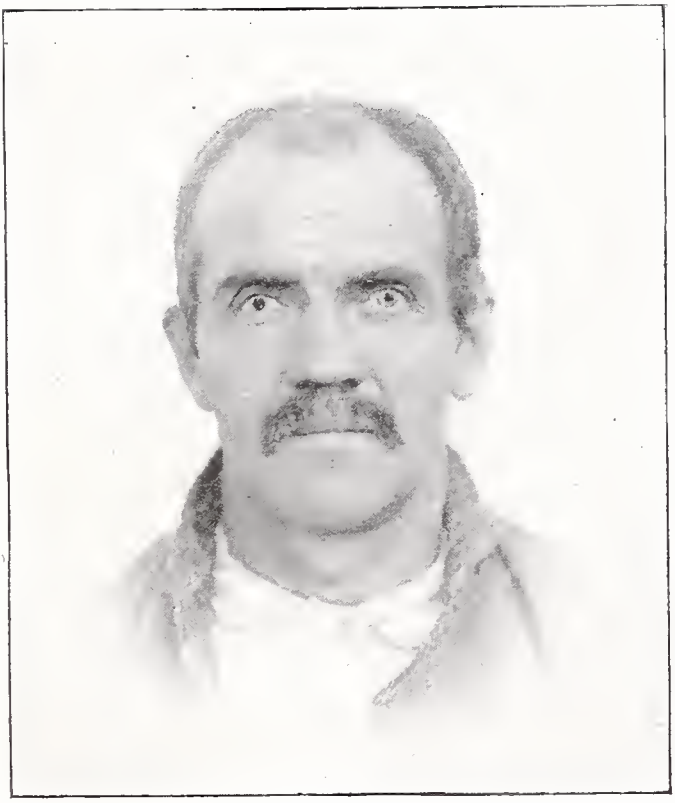
In both eyes there is incipient nerve degeneration. In the macular region of right eye there are a few fine striped points.

In both eyes the retinal veins taper as they enter disc.

The pupils are unequal, the right being larger.

There is paresis of convergence.

FIG. 6.



Photograph showing dilatation of right pupil. Unilateral paralysis of the iris, probably from lesion of the pupillary centre or root-fibres. (Case IV.)

THIRD SERIES.—I. Amyotrophic Lateral Sclerosis or Syringomyelia; Sensory and Trophic Phenomena.—II. Probable Case of Cerebellar Tumor, with Headache, Optic Atrophy, Tetanoid Phenomena and Echolalia and Other Manifestations.—III. Left Hemiplegia Associated with Motor Aphasia in a Right-handed Man.

Reported by E. D. ESTERLEY, M.D., Resident Physician.

Case I.—*Amyotrophic Lateral Sclerosis or Syringomyelia; Sensory and Trophic Phenomena.*

D. C., aged thirty-four, white, weaver, single, admitted January 20, 1894.

Family History.—Mother died of asthma at sixty-four. Father living and well. Two sisters and one brother living and healthy. Two brothers and one sister died in infancy. No nervous history obtainable.

Previous History.—Had measles, whooping-cough, mumps and chicken-pox in childhood. Chews tobacco moderately. Used both whiskey and beer. Denies venereal disease.

About 1885 he began to have a dull pain in right wrist. The arm then grew gradually weaker. In 1887 the left arm also began gradually to weaken, and wasting began about one year later; about same time the fingers of the hand became fixed.

Muscular Symptoms.—Right Arm.—Arm proper seems normal, somewhat small from constant disuse.

Forearm.—Flexors all seem atrophied. Movement at wrist free, somewhat restricted from muscular condition. Can flex and extend fairly well. Lateral movement more restricted, especially on ulner side.

Hand is held open. Thumb lies closely to palm of hand with distal phalanx partially flexed. Muscles of thumb almost entirely gone; hypothenar eminence smaller than usual; a distinct depression in the palm of the hand from atrophy of the interosseous muscles; index finger slightly flexed at first and second joints. Fingers present a bluish appearance; skin somewhat glossy.

Left arm in about the same condition. Flexion at wrist almost entirely negative; can over-extend; thenar eminence gone; distal phalanx of thumb more flexed than right; distinct tremor of thumb; some depression from atrophy in palm of the hand; second and third phalanges of all fingers retain flexed position. They can only partially be extended by force. The index finger responds more readily than the others. A regular "*main en griffe.*"

Dynamometer.—Right, 0; left, 0.

Ankle-clonus present in left and right.

Knee-jerks greatly exaggerated.

Muscle-jerks greatly exaggerated.

Cremasteric reflexes present.

Eye.—Pupils equal and small; respond quickly to light and accommodation.

Station.—Always secure when standing with feet together and eyes closed.

Gait.—Movements of right leg seem abnormal. Left leg is carried out and foot is swung around.

Examination February 10, 1895.—Marked atrophy of both arms, especially so in the ulnar distribution, causing the distribution of "*main en griffe.*" More noticeable on the left side. The interosseous, thenar and hypothenar eminences are wasted. The right hand has not such a marked "*main en griffe*" as the left.

Very active fibrillation in forearm as well marked in the extensors as in the flexors. The muscular nutrition of the arm, shoulder and shoulder girdle is about usual, except some fibrillation about supraspinators and pectoralis major. Muscular condition of legs is normal.

Knee-jerks exaggerated.

Ankle-clonus present.

Sensation.—Some varying areas of anæsthesia, especially marked on abdomen, legs and upper right arm. Thermal tests uncertain; answers are confused, constantly calling cold hot.

Trophic Lesions.—An unhealed eczematous patch on lower angle of left scapula, and similar healed patches over right trochanter and over knees.

No sclerosis.

Case II.—*Probable Case of Cerebellar Tumor, with Headache, Optic Atrophy, Tetanoid Phenomena, Echolalia and Other Manifestations.*

J. B., admitted January 9, 1895, aged twenty-eight, colored, born in Virginia, waiter, married.

Family History.—Not obtainable.

Previous History.—Nothing known. Wife has known him since marriage only, which occurred five years ago; was healthy then, as far as she knew. She has had four children. There was one miscarriage between the first and second child. One child died of meningitis, the second of malnutrition, and the third is living. It is a fine, healthy-looking baby of one year old.

Onset.—About three years ago the patient began to have severe headache. This was paroxysmal in character, and with it the patient would often vomit. This headache would be very severe for two or three days, and would then abate to a considerable extent, although it persisted pretty constantly. There were also periods of vomiting. With this headache he began to complain of dimness of vision, in the right eye first. This gradually became worse, later involving the left eye, and in a very short time this disturbance of vision was so great that he could scarcely get about alone. About a year and a half ago the vomiting became of almost daily occurrence. At this time he was first noticed to throw head back. This position was taken only during the periods of severe headache. The backward position grew to be a more common thing with the paroxysms of headache, and was maintained progressively longer.

On admission, January 9, 1895, the patient came in an ambulance. When he arrived in the ward he was put on his feet. He could stand pretty well, holding

his head greatly retracted; had no apparent impairment of co-ordination. He complained of nothing but headache. He directed his eyes towards examiner when questioned. Some oscillation of eyeballs, possibly due to the blindness. Temperature, pulse and respiration were normal.

Personal Appearance.—A medium-sized, well-nourished colored man, polite in manner, typical African features; facial expression that of a blind person.

Mental Condition.—Memory very poor. Fails to answer questions about time of the onset of the present troubles. Answers questions negatively that should be answered affirmatively. Did not recognize the voice of his wife. Said he had no children. Had great difficulty in interpreting questions. Would repeat question putting it in his own person, and would then attempt an answer, as: "Butler, are you feeling comfortably to-day?" A. "Is I feeling comfortable to-day?" "Let me see, yes." He answered ordinary questions fairly intelligently, but with great effort and after some study. Q. "How did your trouble begin?" A. "How begins?" no answer; then on requesting an answer without repeating the question, he would say that he would do the best he could. "How old are you?" A. "How old is I?" (hesitated) "Will tell you after while." Q. "What month is this?" A. "Let me see. Don't exactly know." At other times he would repeat the question, ask if that was the question asked, and would then proceed to answer it.

Appearance in Bed.—When first placed in bed he had to be restrained, for otherwise he would get up and stand on the bed, and it was feared that he would fall out. His favorite position was on his abdomen with head greatly retracted, back arched to some extent, and legs lifted up also slightly. This gave a peculiar appearance to the opisthotonos, the patient being turned over. The extreme retroflexion of the head greatly exaggerated the position. The shoulders and chest were entirely free from support. Face directed upward, as if in supplication. Arms held under him and hands clenched.

Station.—Fairly steady.

Gait.—Somewhat staggering and weak. Slight pitching toward the right.

Plantar Reflexes.—Right, normal; left, normal. Cremasteric, normal. Knee-jerks—right, subnormal; left, slightly exaggerated.

Sensation.—No areas of anæsthesia, but patient was quite hyperæsthetic. The slightest touch of the pin caused patient to move away, and would set the adductors of the legs into contraction. Tactile sensation seems acute all over the body. No thermal anæsthesia. Very sensitive to cold and heat immediately on application of bottles. Assisted in the examination by counting more intelligently than the average patient. Often stopped and complained of headache. No history of convulsions.

Eye Examination by Dr. de Schweinitz.—Absolute optic atrophy of both eyes. Both sets of vessels contracted, the arteries markedly so. Centres of discs filled in. Some haziness of the margins, probably indicating a pre-existing neuritis; pupils semi-dilated and fixed; slight ptosis of right upper lid.

February 1, 1895.—Retraction of head persisted for about three weeks. Complains of constant headache. Has vomited several times. For a few days was almost in a typhoid condition. Could not swallow. Hearing became greatly impaired.

February 19.—Patient has steadily improved. No retraction of the head for three weeks. Again takes nourishment readily. Can once more feed himself.

Makes known all his wants. Was taken out of bed to-day. Talked more intelligently than at any time since admission. Can now give a disconnected account of himself; seems happy and contented.

CASE III.—*Left Hemiplegia Associated with Motor Aphasia in a Right-handed Man.*

H G., aged thirty-three, white, born in Philadelphia, laborer, single.

Family History.—Negative.

Previous History.—At the age of seven had typhus fever, which left him with a very bad heart. His physician at that time advised his mother not to allow him to over-exert himself, and to have him take up later some light occupation.

Onset.—Patient was among the New Year's shooters, drank a good deal and lost sleep for several nights. He returned to his work, but did not exactly feel well. While seated at the supper table on the evening of January 10, 1895, he fell suddenly from his chair and became unconscious.

On admission, January 12, 1895: A young, pleasant-faced, well-nourished man. No evidence of injury. Scars over nape of the neck showed that attending physician had cupped him; patient could not move left arm nor leg; pupils were equal and about normal in size; protruded tongue did not deviate from median line; mouth was drawn markedly toward the right; patient was conscious and looked inquiringly at the doctor's brass buttons; could say only "yes" to all questions asked, and did this irrespective of the character of the question.

Heart examination showed strong impulse. Murmurs were heard with systolic and diastolic sounds at the aortic cartilage, and with systole at the apex transmitted into the axilla.

Pulse was full, strong and bounding, but very irregular.

Examination of the urine was negative; no loss of control over sphincters. For the first five days the patient was not able to write, later could write his name. The aphasia persists.

The patient is *right-handed*, both according to his own statements and those of his friends.

Eye Symptoms.—Pupils were equal on admission and about normal in size. At 8 P. M. the left pupil was widely dilated.

January 13th, 8 A. M.—Both equal again and normal in size; respond quickly to light. 8 P. M., left pupil about as usual and right pupil widely dilated. This see-saw alternating in equality kept up for about one week. Ophthalmoscopic examination showed nothing abnormal.

Motor Symptoms.—Left arm and leg paralyzed; responds to prick of a pin; endeavors to ward off by use of right foot; protects left arm with right, and changes its position by using right hand. Knee-jerks and muscle-jerks are slightly increased on both sides; ankle-clonus present on right side.

January 14th.—Patient has rested comfortably; temperature, pulse and respiration tend to fluctuate. Moves left leg when pricked by pin; takes nourishment well. Can say only "yes." Looks about the ward as if interested. Endeavors to make his wants known.

January 19th.—Tongue protruded, deviated very markedly to left. This condition persisted, however, only for a few hours.

February 1st.—Thus far has been able to say only "yes." Can write very well. Takes great interest in the affairs of the ward.

February 12th.—Patient continues to improve.

February 16th.—Counted fairly well up to twenty to-day. Surprised me by saying, "Yes, sir, Jesus Christ." Can say "father." Speaks a great many words, but cannot pronounce distinctly. Has improved so much that permission has been given to him to get up. Walks with characteristic hemiplegic gait; left leg is swung around; left arm is carried at side, partially flexed; shoulder on this side droops quite perceptibly.

February 17th.—Patient can say "bread, butter, milk, coffee," etc. Still does not enunciate clearly.

FOURTH SERIES.—Five Cases of Disseminated Sclerosis.

Services of Drs. F. X. DERCUM and CHARLES K. MILLS.

Reported by W. S. HITSCHLER, M.D., Resident Physician.

Case I.—I. H., aged thirty-six, white, Philadelphia, no occupation, single.

Onset.—As far back as the patient can remember she has been delicate and has had some weakness in her legs. Up to the age of eight or nine this was not very marked, and she could run about with her playmates. The patient cannot tell just when the loss of power in her legs commenced, but it came on very insidiously and progressed gradually, so by the time that she was eight or nine years old she was unable to run. In the meantime her arms were losing power almost equally with her legs. She remembers that even at an early age she was troubled with attacks of dizziness.

Previous History.—Had typhoid and diphtheria together when five years of age. She does not remember whether the weakness in the legs was present previous to that time or not. After the attack of typhoid and diphtheria the loss of power in the legs came on gradually. She did not go to school until she was eleven years of age on account of her delicate health. When she reached the age of eleven she seemed to get stronger. Apparently no history of syphilis.

Family History.—Father living and well; mother was sent to insane asylum at Norristown for melancholia in February, 1894.

Symptoms: Tremor.—Came on very gradually, so gradually that she cannot fix any definite time for its beginning. She first noticed it when twenty-seven years of age while knitting and writing, the tremor being volitional entirely. A slight tremor of the head was noticed about four years ago, but it has since disappeared. The legs were never affected.

The trunk partook of a volitional tremor, having to lean the body against a support in order to keep quiet. The body sways now on attempting to sit upright. The tremor of the hands has varied, being better and worse at times; it is very fine when present. At the present time there is no tremor of the hands, but on attempted coarse movements of the hands there is much ataxia. When knitting or crocheting a fine tremor develops, but it does not interfere with the knitting. After she knits awhile the tremor disappears. She has no particular trouble in eating.

Speech.—Her speech has been affected for the past fifteen years. She speaks slowly, and several words are spoken before an abnormal interruption occurs. Then there is a slight tremulousness on the first syllable of the next word, after which she again articulates a number of words quite plainly and evenly, but slowly. There is a slight tremor of the lips on talking. The pitch varies.

Nystagmus.—This was first noticed seven or eight years ago. Is worse in the morning. It is present both on fixation and intended ocular movements. It is lateral and the degree varies, the arc being larger at first and then diminishing. It is sometimes absent.

Paresis.—It has been increasing pretty steadily. There are occasional periods of remission lasting from three months to a year. At the age of twenty-three, while walking across the room, she tripped and fell upon her buttocks. After this accident the difficulty which she had previously experienced in walking was increased, her knees being much weaker. For the last ten years she has been unable to walk at all without assistance.

There is distinct loss of power in both upper and lower limbs and also in the trunk. She cannot stand alone. The power of flexion and extension in the legs is very much impaired.

By the dynamometer the right hand registers 20 and the left 25.

Spastic Symptoms.—None noticed anywhere.

Reflexes.—Knee-jerk, muscle-jerk, flexor-tendon, ankle-clonus, Westphal, planter, biceps and triceps all absent. The abdominal reflex is present.

Pupils respond to light and accommodation.

Gait.—Is not able to walk.

Sensation.—In both legs there is blunted sensation both as to touch and pain, and there is also some perversion. She calls mild pressure with a pin-point a touch of the finger, etc.

On the right leg two points are recognized when placed in the long axis of the limb and over eight inches apart; on the left leg four inches. She cannot recognize two points at all when placed transversely on the legs.

There are also irregular patches of anæsthesia over the backs of both feet. Sensation to pain and touch seems unimpaired elsewhere.

The temperature sense is delayed in the legs, and especially in the feet. Often, but not always, she mistook cold for heat and *vice versa;* after repeated experimentation, however, she recognized them with greater precision.

She never had any girdle pains, but she has had shooting pains from the knee to the ankle and from the waist to the knee three or four times a month for the past five or six years. These pains are not at all severe.

The ataxia in her hands is marked.

Excepting that when her feet are placed together she imagines that they are about five inches apart, there is no defect in the muscular sense.

Eyes.—The pupils are dilated considerably. When they first became so she does not remember. Reaction to light and accommodation is unimpaired.

There is no diplopia nor achromatopsia. Excepting that she is near-sighted her eyesight is good. One peculiarity about the pupils is that they change in size while the eyes are at rest and there is no change in the light. Her senses of hearing and of smell are unimpaired.

Cerebral Symptoms.—Her intellect apparently has not suffered to any appreciable extent. She is pleasant in her conversation and manifests an interest in whatever is said. She is happy and contented. Her memory for remote events is better than for recent events, but this is not at all marked. There have been no apoplectiform or epileptiform attacks. Vertigo is more or less present all the time. No attacks of sudden coma.

Sphincters.—Not affected.

Trophic Disturbances.—For the past four or five years there has been considerable wasting from the hips down.

The little finger of the left hand has a tendency to lap over the third; this was not noticed until her attention was called to it at the present examination. Four years ago she fell and dislocated the first metatarsal-phalangeal joint of the right hand; it was not properly treated, and the result is that the first phalanx is beneath the metatarsal, causing the proximal phalanx to be extended. The other fingers are also slightly extended. There appears to be some slight contraction.

Cranial Nerves.—The only condition noticed is a slight difficulty in swallowing.

Remissions.—There have been occasional periods of remissions lasting from three months to a year. The general course of the disease has been very slow.

CASE II.—J. D., aged sixty-eight, Ireland, single, sailor.

Onset.—During an earthquake in South American waters in 1877, while aboard a vessel, he sustained a great fright, but suffered no physical injury. Until that time he was in good health. About an hour after the earthquake he began to tremble over his entire body, and this trembling has continued up to the present time.

The tremor was so violent, and he was so prostrated from the fright he had received, that he immediately took to his bed. He was in bed for four months in South America, and was then removed to Brooklyn, where he remained for another period of four months. Whether or not his speech was affected at the time of the earthquake he does not remember, but he is certain that he noticed a change in his speech before he left his bed. There was no loss of power at that time.

He has been at the Jefferson Hospital in Philadelphia four times. He has had distinct remissions in his disease, these remissions lasting a year or two. On getting worse he would seek a hospital, where he would gradually recuperate.

Family History.—Knows nothing except that his parents are dead.

Previous History.—Had "Chagres" fever in Central America in 1850; malaria in Virginia in 1863. In the interval he was perfectly healthy. Denies specific history. Moderate drinker.

Present Condition.—*Tremor* of almost the entire body very prominent, the arms and face particularly so; it is coarse and is distinctly aggravated on voluntary movement, interfering much with eating and drinking; he often is compelled to use his fingers in conveying food to his mouth, and even this method is fraught with considerable difficulty and much unsteadiness. Mental excitement also augments the tremor. The tremor is often present when he is attempting no motion whatever, but is not nearly so marked. The tremor is not nearly so pronounced now as it was in the beginning.

Speech is trembling in character, similar to a stutter, in that the sound of the first letter or syllable of a word is repeated before proceeding further. He speaks slowly and the articulation is fairly plain. There is no special tremor of the tongue except that it seems to partake of the tremor of the head.

Nystagmus.—None present, and, according to patient's statement, never was. The left pupil is much contracted. Both respond to light and accommodation.

Paresis.—Began six or seven years ago. It was first noticed in the legs and arms, particularly the former. He walks in a very unsteady manner, taking short steps and grasping chairs, etc., for support (November, 1894).

At present, January, 1895, the paresis is not as marked as when examined in last November. There is much more power in the legs, and he can walk pretty well without assistance. In the same period of time the power in his left hand, as measured by the dynamometer, has increased from 75 to 100, and his right hand from 75 to 90.

Until he came to the hospital his paresis was steadily increasing, there having been no remissions.

There is some spasticity present in the left arm, and some rigidity in the right leg. Passive motion is easy except in the members named.

Reflexes.—Knee-jerk, slightly plus; muscle-jerk, absent; flexor-tendon, absent; clonus, present; cremasteric, absent; Westphal, absent; plantar, present and probably plus; abdominal, present; biceps, present; triceps, absent; iritic reflex, present.

Gait.—Outside of a very slight shuffling movement there is nothing in evidence.

Sensation.—Except in the thighs and legs the sensation is not blunted.[1] At different times in the past twelve years he has had pains in the back, chest and shoulder; they are sharp and shooting in character. A porous plaster, he says, promptly relieves them. No girdle pains at any time. Neither are there any symptoms of ataxia. He has had "pins and needles" sensations at various times, but these are rather indefinite. His sensibility to heat is somewhat defective, evidenced chiefly in the legs; and to cold it is normal, excepting in the legs, where it is considerably blunted. Muscular sense is good.

He can recognize two points three-fourths of an inch apart on the back of the hands, three-eighths of an inch on the forehead, one-half inch on the face, two inches on the chest, four to five inches on the right leg (less than that he regards as one point and refers the sensation to the upper point), five to six inches on the left leg (ditto), two and one-half inches on the left thigh, two and one-half to five inches on right thigh; abdomen, one and one-half inches vertically, four inches horizontally.

Eyes.—No nystagmus, no achromatopsia. Needs glasses to read and to walk at night. There is diplopia at times—horizontal. Objects seem nearer with left eye than with right.

Ears.—Cannot hear the watch tick when held close to either ear. This defect in hearing was not noticed by him until his attention was called to it. He had always supposed his hearing good.

The sense of smell is normal.

Cerebral Symptoms.—There is some mental debility. He cannot think so well as formerly, and he is not nearly so bright. His memory is failing. He cannot remember recent events as well as remote ones. He says he feels content and happy as a rule. No apoplectiform attacks nor epileptiform attacks. He has marked attacks of vertigo which occur in paroxysms. They have been very prominent during these last eighteen months. Whenever he holds his head back, as when being shaved, the vertigo asserts itself. No attacks of coma.

Sphincters.—Not affected.

Trophic Disturbances.—None. The upper teeth are decayed more than the lower, but he says he used to chew glass for bravado. There seems to be no affec-

[1] Vide neuritis attack.

tion of the cranial nerves. There have been remissions in the course of the disease, coming on chiefly in the summer time. The disease has been slow in its progress.[1]

Examination of Eyes by Dr. de Schweinitz.—The pupils are dilated unequally—right, 5 mm.; left, 3 mm.

O. D.—Disc discolored decidedly; veins and arteries nearly the same size.

O. S.—Similar conditions on this side, but the disc is better colored than on the right side. There is a large patch of choroiditis in the lower and inner portion of the eye-ground. There is no paralysis of the external ocular muscles and no nystagmus.

There is a partial gray degeneration of each disc. There is no marked contraction of the field.

CASE III.—B D., aged forty-eight, white, Ireland, married, housework.

It is impossible to obtain much information of this patient, as she is very stubborn and absolutely refuses to submit to an examination.

Onset.—It is impossible to learn when the tremor first began; she does not remember, but it is probably of ten years' duration. She has had some hesitation in her speech for several years. No history of syphilis or alcoholism could be determined. The tremor and difficulty in speech have increased during the years she has been in the hospital. She is exceedingly sensitive and nervous, and attributes her disease to trouble.

Motor Disturbances.—No definite paralysis of the upper or lower extremities determined. Her grip in both hands and power of resistance and movement in arms as tested by duplicated movements are good. She has plenty of strength in her limbs, but her movements are irregular. When observed, she walks with a somewhat irregular, forward, jerky movement. She says that she can walk better when no one is looking at her. When sitting quietly her head is in almost constant coarse oscillation.

The movement affects her whole trunk; this is most noticeable on standing, when the movement of her head also increases.

When at rest and not under examination, her arms and hands are but very slightly affected. There is no tremor in the feet and legs when at rest, but on extending them a slight tremor is noticeable.

All these movements are much aggravated by an effort, or by thinking about them or when observed. There is no tendency for the body or limbs to assume a fixed position excepting a slight one of the head to be tilted backward by the tremor.

Speech.—Her speech is very markedly affected. It is hesitating, syllabic or staccato—*i. e.*, there is a distinct interval between every syllable, although at times a few syllables are pronounced with more rapidity than in her ordinary attempt at talking. For example, she speaks as follows: "I—can—make—beds—all—the—day long," etc.

She seems to speak with difficulty and like a series of slight verbal explosions.

The pitch is the same, as a rule; sometimes under excitement it raises a little.

[1] In October, 1894, he took a sudden and severe chill, which was followed by fever, and which was accompanied by stiffness and great pain in the thighs and legs. The pain was described as "tearing the bones from the flesh;" it was made worse by motion. Excessive tenderness was present. He was two weeks in bed. He became somewhat better by that time, although his weakness lasted some time. Since this attack the loss of power has been more marked.

There is no fibrillary tremor of the tongue, of which she seems to have good control; still, in its upward movement, there is some little tremor and inco-ordination.

CASE IV.—H. W., colored, aged fifty, single, laborer, Philadelphia.

Onset.—When about six years of age, he sustained a severe fright in a steamboat collision on the Delaware river. A short time afterward the tremor began which affected the entire body. This tremor was constant at its onset, but became volitional in the course of a couple of months.

When about eight or ten years of age he began to notice a weakness in his arm and a still greater weakness in his legs, and that the tremor again became constant.

He cannot remember the time that he could talk plainly, but is of the opinion that his speech was affected ever since the collision.

There is no nystagmus.

His intellect has been failing only for the past six months. His bladder and bowels were affected quite early in his disease, but medicine has, according to his statement, greatly relieved this condition.

He has had, at various times, attacks of dizziness and coma. Two remissions have been noted, during the first of which the tremor would cease unless he attempted voluntary movement.[1]

Family History.—Father died of phthisis (?); mother living and well; had no brothers nor sisters.

Previous History.—A few months before the collision he had an attack of scarlatina. He has had chronic rheumatism and pneumonia. Denies both alcoholic and venereal history.

Present Conditions.—A "sere and yellow leaf" old man, his body bent forward with his head looking toward the ground and his eyes elevated.

Tremor.—There is an almost general constant tremor, coarse in character, affecting the entire body. It is most prominent in the lower jaw, where it is exceedingly marked; and then in order in the head, tongue, hands, arms and legs—the tremor in the legs being exceedingly faint. The trunk is not involved.

The tremor is worse on mental excitement and attempted voluntary movement. In the hands it almost ceases on quietude, and to a less extent is this noted in the jaw and head. The motion of the head is rather to and fro than in a vertical direction.

He succeeds in conveying food to his mouth in a manner that seems remarkable considering the intensity of the tremor. With the exception of the two remissions, this constant tremor has been present ever since the accident. During the first remission, which began a few months after the accident and which lasted until he was eight or ten years of age (two to four years), the tremor was volitional only; but during the second, when about thirteen years of age, the tremor disappeared entirely for a period of two years.

Speech.—Is very slow and hesitating and excites additional tremor of lips and tongue. When he desires to speak it often takes him from ten to thirty seconds to utter the first word. After he is once started, however, he proceeds with much greater facility. But there is a pause between each syllable. Often the vowel

[1] In speaking of the various events of his history he will not refer to his age but to his height, by holding his hand a certain distance above the ground.

sound is prolonged, as in saying "ye-e-e-es si-i-ir." There is some little inflection to the voice.

Nystagmus.—According to the patient's statement there never was any. There is none present now.

Paresis.—The paresis came on when about ten years of age. It first showed itself in the arms and then in the legs, the arms being the more affected. It steadily progressed and is quite marked at present.

By Dynamometer.—Right hand, 30; left hand, 35. The patient is right-handed.

Spasticity.—Present to some extent in the upper, but most marked in the lower extremities. There is no marked rigidity of the muscles. Passive motion of the legs is rather difficult.

Reflexes.—Knee-jerk, much exaggerated; muscle-jerk, much exaggerated; flexor-tendon, absent; ankle-clonus, absent; Westphal, absent; plantar, present but faint; cremasteric, abdominal; biceps and triceps, absent. Pupil does not respond to light. Unable to make a statement regarding accommodation to distance.

Gait.—Is spastic-paretic in character and seems to partake of the tremor. The steps are short and unsteady, often grasping at chairs and other objects for support. His walking has improved considerably since last November; a cane which he was compelled to use at that time he has since discarded.

Sensation.—To pain, is slightly blunted in right leg and lower portion of right thigh.

Since his admission to the hospital he has complained of girdle pains. These are not severe. There is not much ataxia, and no formication or other abnormal sensation. The temperature sense is normal. Sensation to touch likewise. The muscular sense is intact.

Examination of the Eyes by Dr. de Schweinitz.—Arcus senilis marked. Pupils are contracted and apparently do not respond to light. The examinations regarding accommodation for distance were exceedingly unsatisfactory. No diplopia.

O. S.—Disc very pallid in the deeper layers. The arteries and veins are about normal in size, although the upper temporal vein is more dilated than the other branches. No nystagmus.

O. D.—Sightless. Tension, No. 1. Lens, cataractous. Cornea is hazy.

Ears.—With the left ear he can hear a watch tick at a distance of one inch from the meatus. Cannot hear with the right ear at all.

The sense of smell is intact.

Cerebral Symptoms.—There is considerable mental enfeeblement. He thinks slowly, but comprehends fairly well. His memory for recent events is poor and for remote events but fair. It is evident, however, that some of his statements cannot be relied upon, and this seems to be the result of his defective memory and not due to any wilful intention to deceive. He feels happy and contented and does not complain. He has had a few apoplectiform attacks, also a few epileptiform attacks, in which both sides were affected. Paroxysmal attacks of vertigo have been noted.

Sphincters.—At present there is partial loss of control of both bladder and rectum.

Trophic Disturbances.—There has been no wasting. He sweats considerably, more so than can be accounted for by environing conditions. Nothing abnormal

has been noticed in the growth of the hair or nails. No herpes. His joints have swelled at times. (He has had chronic rheumatism.)

Cranial Nerves.—He used to have some difficulty in swallowing, but this has since disappeared.

Remissions.—*Vide* ("Tremor.") Whether, during the two remissions, there were changes in any other symptom than the tremor, could not be ascertained.

General Course.—Very slow. His nutrition does not seem much disturbed.

CASE IV.—*Dissiminated Sclerosis.*—R. W., aged forty-two, white, Nova Scotia, single, druggist.

Onset.—About seven years ago he had an attack of coma which lasted half an hour. (He is unable to state whether its onset was sudden or gradual.) Several months afterwards he had sharp pains in the legs, especially at the ankles, which were accompanied by distinct loss of power.

Five years ago he noticed some incontinence of urine. Subsequently, he had an attack of diphtheria, after which the incontinence of urine was aggravated, and in addition he had incontinence of fœces. The incontinence of fœces was not complete, but it forced him to go to stool six or eight times daily to avoid dribbling.

Shortly after this he noticed a numbness in his feet and legs, and also a tremor; the tremor was intensified on attempting to walk. He also noted a failure of memory for recent occurrences, and he became subject to paroxysmal attacks of vertigo. His eyesight also began to fail, the condition being right hemianopsia. His speech was not affected, although in talking it often happened that his memory could not supply the words. Remissions have been noted.

Family History.—Parents, four brothers and three sisters all living and well. No hereditary history in the family.

Previous History.—Scarlatina, measles, whooping-cough and mumps in early childhood; diphtheria five years ago, just previous to the appearance of the chief symptoms of his present disease. He was a constant drinker for twelve years, taking several (?) glasses of whiskey and beer daily, but ceased drinking ten years ago. He had syphilis in 1880, with marked secondary lesions; the only tertiary lesion was an ulcer on the tongue which lasted five years. He was under treatment for this affection but was not thoroughly cured. No history of rheumatism or malaria.

Present Condition: Tremor.—The tremor was almost solely confined to the legs. He describes his heel as striking against the floor in taps while sitting in a chair. The tremor was coarse and constant. It began shortly after his attack of diphtheria and continued for three or four years; it then ceased being constant, and was developed only on attempted movement. It was made worse by occupying certain positions—*e. g.*, sitting with the legs at ninety degrees to the thighs. It was not so prominent when he walked; it would cease when the weight of the body came upon the heel, but it would become intensified when the weight of the body came upon the toes. Movements of the heel would not increase its intensity. The tremor was always increased on excitement.

Since September, 1894, the tremor has been entirely absent, voluntary effort and excitement both failing to develop it, excepting a slight oscillation of the tongue, which occurs on protrusion.

The arms and legs were never subject to a tremor.

Speech.—Not affected.

Nystagmus.—None.

Paresis.—There is considerable weakness in the legs at present, although they are much stronger now than they were three months ago. The arms and hands are not affected. With an occasional exception he has had no erection for eight years; and when it did occur it was feeble and of short duration.

By Dynamometer.—Right hand, 84; left hand, 78.

Spastic Symptoms.—There is some slight rigidity in the left leg. Passive motion is easy.

Reflexes.—Knee-jerk, plus; muscle-jerk, present but faint; flexor-tendon, absent; no clonus; Westphal, absent; plantar, cremasteric and abdominal, present; biceps, absent; triceps, present; tendon-jerk, absent.

Pupils respond to light and distance.

Three years ago the following notes were made:

Knee-jerk, minus; muscle-jerk, absent; no ankle-clonus.

Gait.—Slightly spastic, the right leg being thrown out somewhat in the manner of a hemiplegic. It is constrained and awkward, and the legs are stiff. The right foot used to drag a great deal, wearing out the shoe at the toe. There is not much flexion at the knee.

Sensation.—Tactile sense is blunted from the waist down. He had an attack of erysipelas in the legs about three years ago, and since then there has been distinct numbness and insensibility in those members. There is no blunting of sensibility above the waist. Muscular sense is unimpaired. Standing with his eye closed his position is unsteady.

Special Senses: Examination of the Eyes by Dr. de Schweinitz.—Quadrant hemianopsia.

O. S.—The pupil is normal in all respects. The disc is irregularly oval; there is a shallow cup making the temporal half pallid. The disc is nearly round with a slight excavation; there is a green tinge to the upper and inner border, but to a less extent than the right eye. The sensory reflex sharply marked on the temporal side. Vessels normal. Pupils equal. Irides alike but more homogeneous than usual. There is no diplopia now.

O. D.—The sensory reflex is sharply marked on the temporal side. The upper inner quadrant of the disc presents a distinctly greenish hue. The vessels are about normal in size. Macula normal.

The above notes were made July 22, 1892. The following on November 26, 1894:

O. D.—Good color except directly outward, where there is a distinct triangular patch of atrophy.

O. S.—Similar, except that the atrophic patch is up and out, and there are no other changes in the eye-ground. Nystagmus is not present.

Ears.—With the left he can hear the ticking of a watch two feet distant; with the right he cannot hear at all.

Smell.—Seems to be intact.

Cerebral Symptoms.—Excepting loss of memory there is no mental weakness. His memory is good for remote, but poor for recent events. Proper names and words that are ordinarily used in conversation, but with which he is perfectly familiar, he cannot remember easily. He has had two or three apoplectiform attacks. The loss of power in each case was slight and gradually improved. He does not remember having any attacks which simulated epilepsy. The paroxysmal attacks of vertigo ceased two years ago. He is contented. There is no dementia, melancholia nor exaltation present. He is quite apt in conversation, and his intelligence

does not seem to be impaired in any way. Note in 1891: "He is apathetic as to current and public events, and his interest can be aroused only with difficulty."

Sphincters.—As was noted above, he lost control of his bowels and bladder five years ago, and in this respect he has not greatly improved. At times there was some improvement, but it was not marked, nor did it last long. He is compelled to wear a urinal.

Trophic Disturbances.—There is no wasting, but on the contrary he is gaining in weight. His hair has been falling out, but this may be attributed to syphilis. There have been no joint affections, nor nothing abnormal in the growth of the nails, etc.

Cranial Nerves.—Nothing noticed.

Remissions.—Duration three or four months.

General Course.—Has been slow.

FIFTH SERIES.—I. Malarial Multiple Neuritis.—II. Cerebral Abscess, Involving the Internal Capsule, Lenticula and Thalamus.—III. Softening of Lenticula and Internal Capsule; Three Foci of Softening in the Right Temporal Lobe; Pontile Cyst.

Services of Drs. WHARTON SINKLER and CHARLES K. MILLS.

Reported by E. A. SHUMWAY, M.D., Resident Physician.

Case I.—*Malarial Neuritis.*

J. W., aged thirty-six, white, domestic, married. Both parents died of old age.

Personal History.—Usual diseases of childhood; small-pox and typhoid fever at about twenty years of age. Has been married eighteen years; seven children; three miscarriages at from six to seven months. The other four children born living. Three died before two years of age from "summer complaint," etc. One child living and well at three years. No alcoholic or specific history obtainable.

Had always been in quite good health until April, 1894, when she began to have chills and fever. The chills came usually in the afternoon, and were severe, lasting from one and one-half to two hours, and were followed by high fever. These attacks had lasted for about four weeks, when she was brought to the hospital, much reduced in strength and markedly anæmic. She had a number of well-marked chills in the medical wards after admission, the temperature reaching 105° F. The *plasmodium malariæ* was found in the blood repeatedly, and the chills were controlled by the administration of quinine. She slowly recovered strength under treatment.

June 1st, on attempting to get out of bed, her feet slipped from under her, and she fell to the floor. Examination showed complete loss of power and sensation in both legs below the knee and in the left hand. She attributes this to the use of hot-packs. Tincture of iodine was applied to the fingers.

On June 19th the numbness in the hand and leg had disappeared, and was replaced by tingling sensations. The muscles of the calves were flabby. Power in the legs and left hand was still much impaired; she was unable to grasp anything with certainty. Patellar reflexes were absent. Marked shooting pains began soon in the calves and joints (ankles, knees and metarcarpo-phalangeal joints of the feet and left hand). Well-marked tenderness on pressure was present along the lines of the nerve-trunks; also shown on grasping the muscles. She showed evident hyperæsthesia in the legs below the knees, and in the left arm below the elbow. She was treated with sodium salicylate, Fowler's solution and tincture of nux vomica.

She remained in much the same condition until December 1, 1894, improving at times, and being able to walk around with support, then going back to bed again

to remain there for several days. Since December 1st she has been somewhat better, hyperæsthesia growing less and power slowly returning to the legs and arm. During January she had several paroxysms of malaria, during which the plasmodium was again found. The paroxysms were readily controlled by quinine.

Admitted to the nervous wards, March 5, 1895.

Present Condition—Examination.—Loquacious woman. Fairly well developed, but muscles are flabby, particularly those of the legs below the knees. The left hand shows some evidence of wasting. Both feet are œdematous below the ankle, and the circulation is poor. There is some foot-drop on both sides. Lungs and heart normal.

Motion.—Cannot bring feet to more than a right angle with the legs. Left hand cannot be tightly clenched. Otherwise motion is normal.

Sensation.—Hyperæsthesia of both legs below the knee, and to a less extent the left arm below the elbow. Palmar surface of fingers most sensitive. No areas of anæsthesia. Temperature sense present, although her answers are not always accurate. Pressure sense good.

Position.—Fair, sways slightly.

Gait.—Uncertain; resembles "steppage" gait, though not entirely.

Reflexes.—Knee-jerk, absent; muscle-jerk, absent; ankle-clonus, absent; plantar, increased.

Complains of dull, aching pains in the legs in place of the sharp, stabbing pains formerly present. Pain across the ball of the foot, especially after standing or walking. Hearing, good.

Eyes examined by Dr. Oliver.—Partial degeneration of the optic nerve, retinal arteries being diminished in size; the retinal veins excessively engorged. Fields of vision are reduced to about one-half of normal size. There is no motor disturbance.

Case II.—*Cerebral Abscess, Involving the Internal Capsule, Lenticula and Thalamus.*

I. J., aged twenty-five, single, white, domestic.

Admitted to the surgical wards, September 13, 1894, with necrosis of the tibia, the result of an accident when three years old. While here she showed evidences of mental deficiency with hysterical tendencies, crying and laughing readily. On several occasions she had severe attacks of pain in the epigastric region; these were accompanied by sudden rises of the temperature, and at times with rigidity of the abdominal walls and marked abdominal tenderness. The attacks lasted from two days to a week, and, as they passed off, she would renew her work in the department. She was discharged from the surgical wards, December 3, 1894. (Patient stated that at one time she was in a trance-like condition for five months; she was stiff and cold. She denied alcoholic or specific history.)

Re-admitted to medical wards, January 7th, with a temperature of 102⅖° F., with a coated tongue and constipated condition of bowels. The mental condition was poor; she complained of pains in the head and right side of the neck, from which she had been suffering for several days. Blisters had been applied to the temporal region on the left side before admission. This, she said, had been followed by marked conjunctivitis and œdema of the left eyelid, which still remained, and prevented exposure of the eyeball voluntarily. The head was drawn to the left side.

Two days later (January 9th) the temperature rose to 103° F.; the patient became stuporous and could not answer questions, though conscious of any remark made to her. She lay with her head to the left side, moaning frequently. There was marked tenderness behind the mastoid process on the right side and slight œdema of the right side of the neck. The left eye deviated strongly to the right; right eye deviated to a less extent. Left pupil normal; right contracted. Both reacted well to light. Tongue protruded straight.

Toward evening the left arm and leg became spastic; the left cheek and angle of the mouth drooped, and the tongue deviated to the left side. In both leg and arm there were scattered areas of partial or delayed sensation, in a few places amounting to complete anæsthesia. The superficial reflexes were diminished, but the deep tendon-reflexes were markedly increased.

Examination of the right eye at this time showed œdema of the optic disc and nasal half of the retina. The left eye could not be examined owing to the œdema of the lid. No sign of choked disc was apparent.

The pulse-rate was slow in proportion to the temperature. There was some evidence of lung complication—dulness posteriorly at the base with some moist râles, but no tubular breathing. Urine and fæces were voided involuntarily. The urine showed a small amount of albumin, but no casts were found.

On the tentative diagnosis of gumma of the brain, injections of mercury, one drachm three times daily, were begun.

The patient remained in this condition of stupor for over a week, the lung condition clearing up gradually, and the mental condition improving slightly. Marked œdema and brawniness of the tissue of the scalp were noticeable. On January 18th she began to scream occasionally, and a note on June 26th states that the screaming still continues, being stopped by morphia alone.

On the 30th her condition was much improved. The inunctions were discontinued, owing to the appearance of a diarrhœa. On February 1st there was a discharge of a drachm of pus from the left ear. A trophic lesion developed on the left heel, a larger bleb forming, which was filled with bloody pus. February 20th she had improved very much. Talked rationally. The ptosis had disappeared, but there was no sign of return of power to the left arm and leg.

Transferred to nervous wards March 1st.

Examination, March 4, 1895.—Patient much emaciated, particularly about the face. She seems quite rational, but laughs or cries irritably. Mentality is quite low, and it is difficult to get reliable answers as to sensation. She is quite weak; lies quietly with the head turned usually to the left side. The mouth droops on the left side, and on smiling is drawn upward on the right side. Bed-sore on left heel.

Motion.—Apparently lost on the left side, although the nurses assert they have seen her move her left arm and draw up the left leg when she thought no one watched her.

Reflexes.—Knee-jerks, muscle-jerks and arm-reflexes exaggerated on left side. Plantar reflexes present. No clonus. Conjunctival reflexes present.

Sensations.—Answers are very unreliable. Conduction seems to be everywhere preserved. On the left side there is apparently hyperæsthesia. She complains when touched on that side or when her limbs are moved in flexion or extension. Temperature sensations could not be determined, owing to mental condition. There was marked tenderness along the lines of the nerve-trunks.

Urine and fæces are voided involuntarily. There is a discharge of pus from the left ear. She complains of tenderness over the mastoid process of the right side, but no œdema can be observed. It is very difficult to understand what she says unless she makes an especial effort to make herself intelligible.

Examination of Eyes by Dr. Oliver.—O. D.—Edges of disc everywhere veiled; nerve-head slightly swollen; œdema; retinal arteries and veins normal in size; nasal edge of disc more swollen than temporal; iris is more prompt than in O. S., but reaction in both is obtained from all parts of the field.

O. S.—Disc shows same character as in O. D.—œdema. The nasal edge of the disc is more hazy than the temporal. The retinal arteries and veins are normal in size. O. S. pupil the larger.

March 8, 1895.—Patient remains in much the same condition; she whimpers if disturbed, especially if joints are moved. She is quiet usually during the day, but is very noisy at night, disturbing the patients in the ward. Hyoscin and trional quiet her for a short time, but she soon resumes her crying and fretting.

Treatment has been stimulation—strychnine sulphate, tincture of digitalis and whiskey, with liquid diet.

March 23, 1895.—For the last week has been much weaker, at times seemingly unable to answer. Pulse, very weak; respiration, shallow; swallowing at times difficult; tongue, dry and brown from breathing with mouth open; pupils respond very sluggishly; conjunctival reflexes very inactive. Still noisy at night. Died apparently from exhaustion.

Autopsy.—The dura was found adherent to the skull and for about one-third of an inch to the arachno-pia, across the cephalic extremity of the horizontal portion of the Sylvian fissure, there being slight attachment to the subfrontal and supertemporal convolutions. An irregular layer of pus was seen on the caudal portion of the subfrontal and lower extremities of the pre-central and post-central convolutions, also spattering the superior temporal convolutions. Some pus had found its way either before death or during the post-mortem examination to the fossa along the right halves of the pons and oblongata, giving the appearance of a purulent meningitis in this region. No other external appearances of brain and membranes were observed. Sinuses all examined and found empty.

On opening the right lateral ventricle its floor was found intact, but over the cephalic portion of thalamus, and the caudal half of the ventricular portion of the caudatum, it presented a dirty, yellowish and somewhat sunken appearance. A transection just at the cephalic extremity of the thalamus, at a depth of about 3 or 4 mm., revealed a circumscribed abscess-cavity filled with greenish-yellow pus. Examination of this cavity showed that it had destroyed the mid-regions of the internal capsule, a large portion of the lenticular nucleus and thalamus. It was mainly beneath the caudatum, but a sinus reached forward beneath the head of this body slightly into the white matter of the prefrontal region near the median line. The abscess occupied one of the regions so commonly invaded by an intracerebral hemorrhage, and reached also into the corona radiata.

Case III.—*Softening of Lenticula and Internal Capsule; Three Foci of Softening in the Right Temporal Lobe; Pontile Cyst.*

W. E., aged fifty-three, white, Germany, soldier. Admitted to the nervous wards, February 26, 1893.

Previous History.—Had cholera in 1866, followed by typhus. He was a heavy drinker. Denied syphilitic history.

Two weeks before admission, on coming home from a hard day's work, he found he had lost power in the left leg and left arm. No loss of consciousness nor aphasia.

Examination on admission showed paresis of left arm and leg, which he could move slightly. Tongue, when protruded, deviated to the right. Unable to walk. Sensation was apparently unimpaired. No aphasia nor amnesia present. There was no headache except on lying down.

Transferred to out ward, August 10, 1894.

On March 2, 1895, he was found in a semi-stuporous condition, unable to walk. It was difficult to understand his speech. Arms and legs were paralyzed more completely, and were in a spastic condition, the arm being rigidly contracted at the elbow. He was transferred to the nervous ward the same day, where the above conditions were confirmed. Aphasia was present, but not to a marked extent. Reflexes exaggerated markedly on the affected side. Sensation could not be determined, owing to mental condition of the patient.

Lungs negative. Heart dulness increased in area; mitral systolic and aortic systolic murmurs; urine was free from albumin and sugar.

He was placed at rest in bed and put on digitalis, minims x every four hours, with a milk diet. The patient did not improve. The stupor gradually deepened, and aphasia became more marked, until he could not answer questions addressed to him. Urine was voided involuntarily. He slept but little at night, but complained of no pain. The pulse became weaker in spite of stimulation, and respirations grew shallower, finally becoming typically Cheyne-Stokes in character, the patient ceasing to breathe for twenty seconds out of a cycle of sixty seconds, the remaining forty seconds being occupied by a rise to a climax and gradual fall again to cease altogether. On the 10th the temperature began to rise and fall irregularly, reaching 102° F.; respiration increased from 30 to 70; pulse to 136, and the patient died on March 11th.

Post-mortem Examination.—Lungs, normal; cavities of the heart all enlarged. The right ventricle wall slightly hypertrophied; left ventricle wall thickened about one-half and enormously enlarged in cavity. Muscle is pale and rather soft. The mitral valve shows a distinct thickening and resection of the leaflets. The aortic valve shows a chronic vegetative enlargement in the centre of the margin of each leaflet. There is no ulceration. The coronary openings are free. The coronary arteries thickened and showing patches of atheroma.

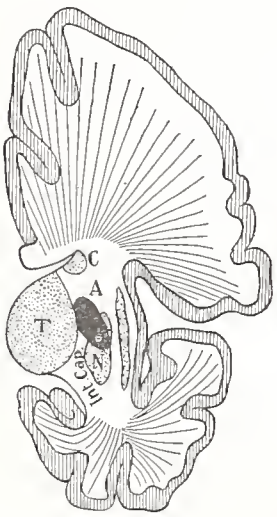
Aorta extremely atheromatous, and shows a large number of calcareous plates. Interstitial nephritis.

Calvarium somewhat thickened and quite dense. Dura mater not adherent. Inferior longitudinal sinus filled with blood. On cutting open the dura there is a free discharge of serum. The pia-arachnoid perceptibly filled with serum. On removing the brain an area of softening is seen on the right side three-quarters of an inch in length, and one-half inch in each of the other diameters is found on an inner extremity of the temporal lobe, and another one on the outside of this one, being separated from it by one-quarter inch of tissue. A third area of softening in the temporal lobe of right side, extending from the horizontal limb of the fissure of Sylvius an inch back of the ascending limb backward and downward a distance of two and one-half inches. The vertebral and basilar arteries are

extremely calcified. The internal carotid, the middle cerebrals and anterior cerebrals are all in extreme atheromatous condition.

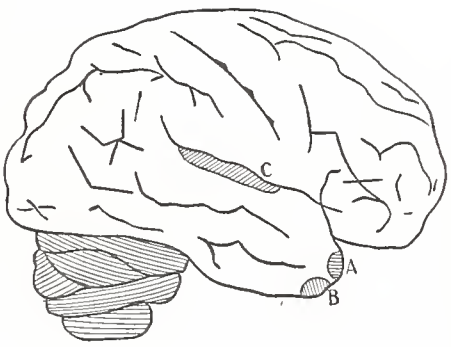
The circle of Willis is normal in formation. The lateral ventricles are both enlarged. The choroid plexus shows a number of small cysts. In the right side of the pons there is a cyst about one-quarter of an inch in diameter. The oblongata and cerebellum show no lesions. Involving the internal capsule and lenticular nucleus on the right side in its posterior extremity is a cyst of softening about one inch in length and one inch in width, and one-quarter of inch in the other diameter. (See Figs. 1, 2 and 3.) The other organs were normal.

FIG. 1.



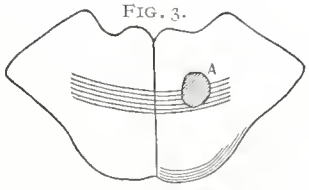
Old embolic cyst involving the internal capsule and lenticula. A, cyst; T, thalamus; C, caudatum; N lenticula.

FIG. 2.



A, B, C, areas of softening.

FIG. 3.



A, area of softening on right side of pons.

MULTIPLE SOFTENING FROM EMBOLISMS. (CASE III.)

TWO CASES OF LOCOMOTOR ATAXIA: ONE IN A CHINAMAN, THE OTHER IN A NEGRESS.

SERVICE OF DR. JAMES HENDRIE LLOYD.

REPORTED BY DR. W. B. IRISH, Resident Physician.

CASE I.—Leong Jung Yang, Chinaman, aged forty-four, married, laundryman by occupation.

Family History.—Patient can give no history which shows any hereditory influences. On account of his inability to understand English, only a vague history of the patient can be obtained. He had rheumatism several years ago. He denies syphilis, but says he had gonorrhœa once. He has used tobacco, alcohol and opium moderately.

Present Disease.—The patient was well and strong until thirty-one months ago, when he first noticed shooting pains in the back, abdomen and lower limbs, the most intense pain being in the legs and feet. These have continued throughout the disease, with the addition of gastric crises. No other history of the disease can be obtained.

Examination of the patients body shows it to be well nourished, with no apparent wasting nor paresis of any of the muscles. Dynamometer registers—right hand, 37; left, 47.

There is a scar in left groin, evidently the result of a suppurating bubo. Other scars over the tibiæ are of a dirty brown color and very likely are specific in origin.

On examination of the eyes some nystagmus is noticed. The pupils are about normal in size, the right being slightly larger than the left. Both pupils respond normally to light and accommodation.

Auscultation of the chest gives slightly roughened breathing at right apex. Otherwise, the lungs are normal. The heart's action is rather weak, but no murmurs are heard.

Knee-jerks, muscle-jerks, front-tap and plantar reflexes are all absent. Cremasteric reflexes are present.

There is total thermal and tactile anæsthesia from the soles of the feet up to within three inches of Poupart's ligament anteriorly, and the same height posteriorly. Another area of anæsthesia, three inches in diameter, is present just over the inferior angle of right scapula. Over the remainder of the body sensation is apparently normal, excepting in the lumber region, where it is somewhat delayed and blunted.

The movements of the patient show a high degree of inco-ordination. For instance, he finds it impossible to button the shirt-front or to touch the tip of his nose, the eyes being closed. In walking he is obliged to use two canes, and then can only do so by constantly keeping his eyes on the floor. The legs are thrown out violently, the feet raised high and brought down with the sole of foot and heel striking the floor simultaneously. The above symptoms are gradually becoming more and more pronounced, with the power of locomotion more difficult.

CASE II.—S. G., aged forty-seven years, black, single, domestic by occupation.

Family History.—The father and mother of the patient were full-blooded negroes; the former died of phthisis, the latter of heart disease. No neurotic history obtainable.

Previous history of the patient shows that she has had the diseases of childhood. She had gonorrhœa several years ago, but specific infection is denied. Patient states that she has beeu delicate all her life.

The present trouble was noticed first about two years ago, when a sensation of general weakness was felt. After this had beeu present for about three months she gradually lost the power of locomotiou, so that in a very short period of time it became impossible to walk without support. This was not on account of the weakness, but because she was unable to retain her equilibrium. About this time she began to suffer with pains shooting through the body. They were especially severe in the legs and feet.

Since admission, thirteen months ago, she has had, in addition to that just mentioned, a frontal and occipital headache, which is now almost constaut.

Examination of the body shows an emaciated negress, who lies quiety in bed (she has been unable to walk for several months), auswering questions intelligently, but not speakiug unless addressed. The limbs are shrunken and atrophied, the feet extended, and the right leg turned outward, half way around. The left knee is swollen (Charcot's) and greatly deformed; the right is somewhat similar, but not to such an extent as its fellow. There is entire loss of power in this limb only, but some paresis is noticed in the upper extremities, as is partly shown by the dynamometer, which registers, right hand, 18; left, 12.

Examination of abdominal and thoracic viscera reveals nothing abnormal.

Pupils are equal in size, contracted, and respond neither to light nor accommodation.

Knee-jerks, muscle-jerks, ankle-clonus, front tap aud plantar reflexes are all absent.

There are no areas of the body where total anæsthesia is present. In the popliteal space, and extending up the posterior aspect of right thigh about four inches, is an area of hyperæsthesia, the sensation, however, being delayed a couple of seconds, Ou the remainder of the limb, and whole of the opposite one, seusation is blunted and delayed at least five seconds. Sensation is apparently normal on other portions of the body. Thermal sense is entirely preserved.

(The above cases are reported as contributions to the study of locomotor ataxia in the various races. It has been claimed that locomotor ataxia is rare, even unknown, in the negro race. Its occurrence iu negroes has certainly been rare in Blockley, for the above case is the only one seen there during recent years. As to the relative occurrence of the disease amoug the Chinese, data are probably wanting. There is no special reason, however, for supposing it to be rare among them. J. H. L.)

A BRIEF RÉSUMÉ OF A COMPARATIVE STUDY OF THE OPHTHALMOSCOPIC CONDITIONS SEEN IN INTERSTITIAL NEPHRITIS AS FOUND IN DISPENSARY SERVICE AND IN GENERAL HOSPITAL PRACTICE.

By CHARLES A. OLIVER, A.M., M.D.

Through the kindness of several of his colleagues, the writer has had the rare opportunity to study a series of ward cases of this type of disease, and thus compare their ophthalmoscopic findings with those that, by long experience, he has become so familiar with in the ordinary walking cases that are so frequently seen in eye-dispensary services.

This difference, which was first pointed out to him by his teacher, Dr. William F. Norris, appeared so radical when he examined his first few cases that he decided that a brief paper upon the subject in these Reports might be of some practical use and value.

Instead of the marked degenerative lesions in the optic nerve-head, retina and choroid, and in place of the gross alterations of the blood-vessels and lymph channels that are so common in the dispensary case, there appeared in every instance, in all manner of varying degrees (mere traces at times), a true neuro-retinitis with vascular and lymph-wall changes.

The ophthalmoscopic picture presented most frequently by the Hospital case is one where associated with hemorrhages, which are most often recognized as flame and fan-shaped extravasations in the thickened fibre-layer of the retina, especially along the main vascular stems, there is nerve-head swelling and œdema from the very faintest haze and elevation to the most complete choking and blocking. In addition to these, just beginning diminution of the arterial currents, which is more marked when the disc is swollen with irregular thickenings and opacifications of the vessel-walls themselves, can be recognized. At times, at points along the finer branches, the walls themselves seem actually thinned and the contained currents bulging. Later, minute yellowish specks and flecks in the deeper retinal tissue and the choroid appear—the entire grouping of symptoms as evidenced through the magnifying power of the direct method of ophthalmo-

scopy, being expressive of a disease which manifests its greatest brunt upon the vascular and neural system—a disease whose series of ophthalmoscopic conditions form merely a part and parcel of what may be made plainly evident post-mortem to the increased power of the microscope throughout the related vascular and neural structures of the entire body.

This picture is totally different from that which is seen in the walking case at the eye-dispensary. In this variety of subject, there is oftimes a subsided neuro-retinitis with scars of frequently repeated past inflammatory action and marks of renewed disturbance. These, with localized degenerative atrophies of the optic nerve-head, retina and choroid, and fatty and granular degeneration areas, are all the prevailing conditions. Here the ocular changes have been less apparent in their various stages, and it is only during the late and incurable period of the general disease that the patient seeks the aid of a medical adviser for failing vision. In fact, they have been more insidious, and hence, in reality, more grave. Again, the dispensary case is the one where the general disease—although, as a rule, having existed for a much longer period of time than the hospital type—has never appeared one of sufficient moment to render the subject bedridden for any extended time, and frequently the history of this class of cases will show that the patient has been but seldom incapacitated from following his usual occupation. This type often comes primarily to the ophthalmologist complaining of defective sight, and, never having been apparently seriously ill, has allowed the early and inflammatory signs of optic nerve, retinal and choroidal disturbances to pass unnoticed.

In the dispensary case the climax of the general disease, as it were, which, as a rule, has never been great, has passed away, and the varying groups of ocular degenerations seen ophthalmoscopically represent in a rough way merely to what extent the recurrent inflammatory reactions have reduced these degenerate tissues and to what degree the existing exacerbations are allowed to proceed.

In the hospital type of case here studied, the condition of affairs is far different. The eye-ground has, by reason of the patient's general state, been allowed either an early examination or one when its disturbance is at its greatest height or during the incipiency of the degenerative process.

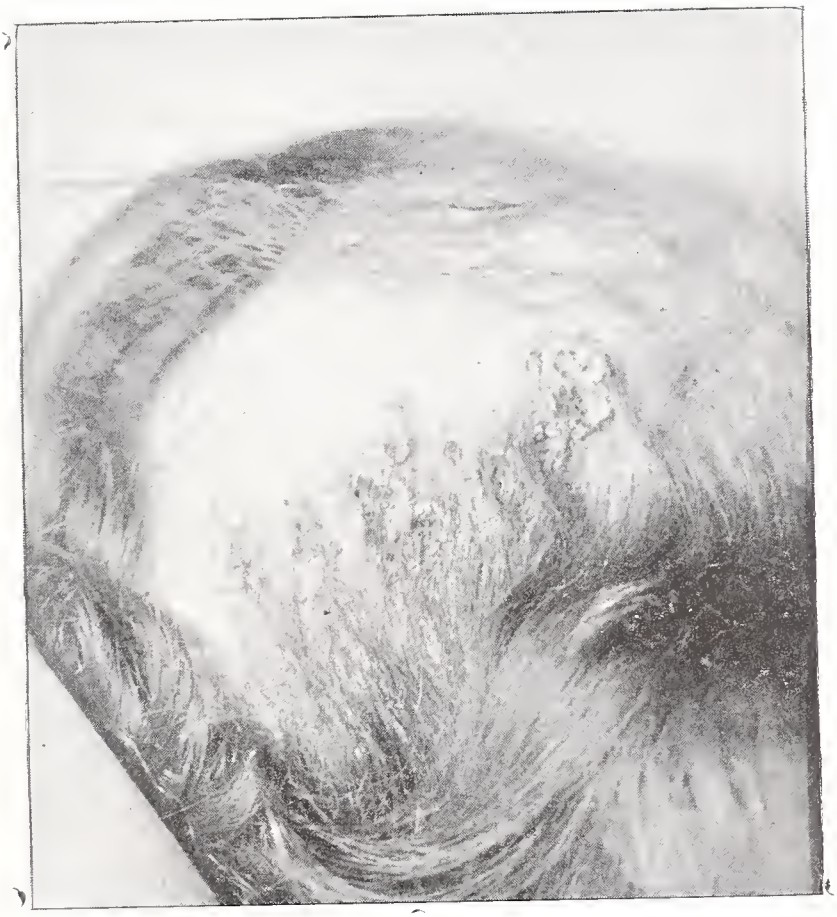
NOTES ON FAVUS.

By HENRY W. STELWAGON, M.D.

In all, sixteen cases of favus of the scalp have been under treatment in the skin wards during the past four or five years. All were cases of long standing, varying from two to ten or more years. In one instance only was the subject a native-born American. Of the others there were five Russians, three Italians, three Germans, two Irish and two Roumanians. Their stay in the hospital averaged about three to four months; no case was discharged entirely cured, however, but the majority were so markedly benefited that a discharge was asked for, and in others it was felt that further treatment could be carried on just as well by the patients at their home, thus relieving the institution of their support. The extent of the disease varied greatly, from an eighth part up to the entire scalp. Atrophic and scar-like changes (Plates 1 and 2) were to be noted in all. In some, the crust formation was slow; in others, it was astonishingly rapid. In some of the cases parts of the scalp at which the disease had appeared originally were now free from active manifestations, the disease apparently having worn itself out, leaving behind atrophic scar-like skin, with here and there hairless areas, hair tufts and scattered hairs.

In one case (Plate 3) reported by my colleague, Dr. Cantrell, in the *Journal of Cutaneous and Genito-Urinary Diseases* for September, 1894, the scalp had been diseased in its entirety for years, and the affection had then invaded the general surface. This case was admitted at the end of my colleague's term, and came, therefore, under my care for treatment. While it is commonly believed that favus of the general surface is readily cured, in which my own experience accords, yet in this single instance at least two months elapsed before the skin was permanently freed from the crusts. A strong sulphur ointment was employed, both by rubbing it in and applying as a plaster; at the end

FIG. 1.



Favus of scalp.

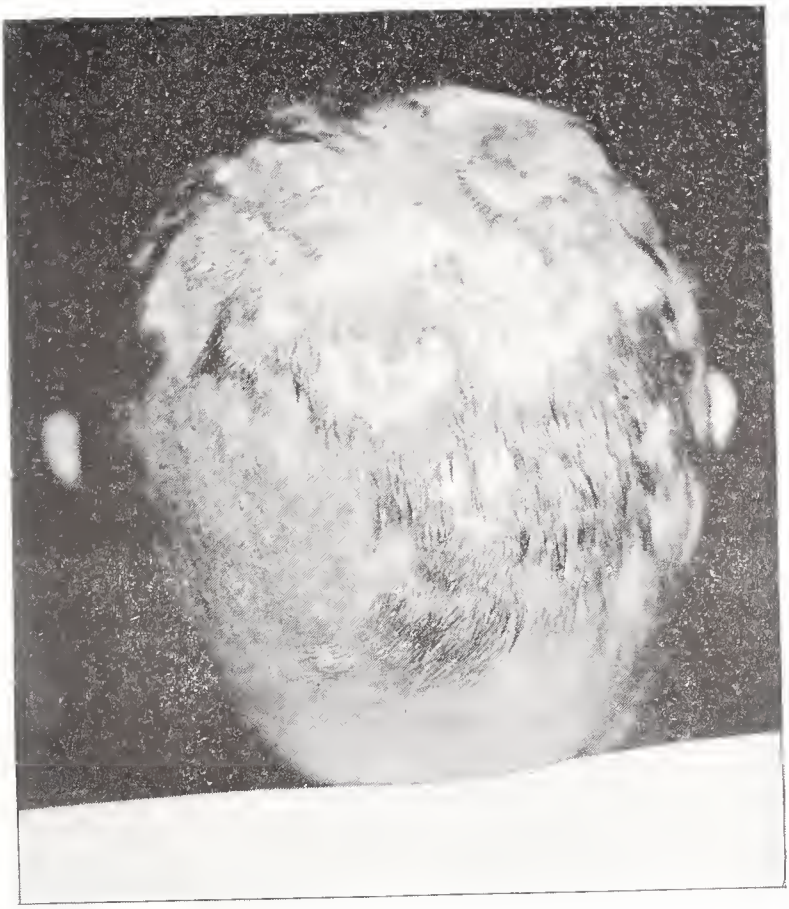
of a few weeks relief was apparently complete, but upon cessation of the baths and application, in several of the areas the peculiar cup-shaped crusts soon began to reappear, and this disposition persisted for almost ten weeks.

Favus of the scalp is a most intractable disease. In many instances it may, indeed, be said to be practically incurable; that is, the time required to bring about permanently favorable results is so long, varying from at least six months to several years, and the measures of treatment so irksome and tedious, that very few patients among the class in which this disease prevails will be found to be sufficiently persevering. It is true the disease after years tends to gradually wear itself out, and patients will occasionally be seen with the evidence of its ravages, such as atrophic scarring and baldness, in whom after a duration of ten, fifteen or more years spontaneous cure has resulted, helped, doubtless, by treatment pursued irregularly from time to time. In my earlier experience in dispensary practice I often thought I saw cures resulting after several months' treatment, and apparently this was the case, but a larger observation and returning patients have taught me that while the disease was always benefited and somewhat diminished in area, a permanent and complete cure requires a much longer period and the employment of actively energetic measures of treatment. In such instances an apparent cure took place, as evidenced by the condition of the skin, the freedom of the hairs, etc., and these favorable conditions would often continue for several weeks or one or two months, the disease then gradually reappearing, usually, as remarked, in area of less extent. Observation has thus shown that a few months' treatment will usually destroy all the readily reachable fungus, the duration of the apparent cure lasting for the period required for the growth of the deep-lying fungus to make its way to the surface. The growth in favus always gets deep down into the cutaneous tissues, and it is this portion, which in part at least escapes destruction or removal, which serves as the seed, so to speak, for a relapse. I am convinced, therefore, from observation of a large number of dispensary cases as well as the cases here under consideration, which latter especially have been most energetically treated, that the so-called cures in three or four months' time are in reality rarely permanent cures; this has been further emphasized by cases which have gradually drifted to my service in making their rounds from one dispensary

to another, as these patients do, in whom the disease, so it was alleged, had been pronounced cured one or more times by other physicians under whose care they had previously been.

As to treatment, many remedies have been employed by various dermatologists, but my own practice has finally led to a choice of one of three remedies—sulphur, chrysarobin and mercurial applications. This conclusion has not been reached without a trial from time to time of other remedies, for which special claims may have been made. Before speaking of these remedies a measure of treatment should be referred to, which is of material, and I may say even essential, importance, and that is depilation. The most efficient plan of depilation is by means of the forceps, going over the whole diseased area, taking a small part each day; it is slow and somewhat painful, and must, moreover, be repeated in many cases two or three times before a complete cure results. It is found, however, that the second or third depilation, if it is required, need not be so general as the first. The application of a strong carbolic acid wash once or twice daily will lessen the pain of this procedure. The hair of the entire scalp should be kept closely cut. Occasional or weekly washing with sapo viridis and hot water should always be advised. As to remedies mentioned—sulphur, mercurials and chrysarobin—the three are almost equally valuable, although, in my judgment, if there is any superiority, it is with the mercurials. Sulphur is applied as the officinal ointment, or weaker if the skin is irritable. The best mercurials are blue ointment and corrosive sublimate lotion; the former pure or weakened, and the latter in the strength of one to four grains to the ounce of water. Chrysarobin is applied in an ointment, thirty to sixty grains to the ounce. Whatever ointment is employed it should be applied by rubbing it thoroughly into the skin, and then, if possible, further applying it as a plaster, spread upon lint or any other suitable material. All these ointments cause, after a time, more or less irritation, and must be suspended temporarily for two or three days, during which a lotion of boric acid or an exceedingly weak ointment of sulphur may be employed. With the use of blue ointment care must be exercised, and the slightest evidence of absorption be a sign for its temporary withdrawal; this has, however, rarely been found necessary unless the disease involved the entire scalp. Corrosive sublimate lotion I have used freely in a number of cases, two and even three grains to the

FIG. 2.



Favus of scalp.

FIG. 3.

Favus of general surface.

ounce, and applied it to the whole scalp, and beyond the local irritation sometimes caused, no untoward effect has been noticed; the possibility of toxic action should, however, not be lost sight of, and if the diseased area is extensive, a weaker lotion should be first employed. Even when the entire scalp is not involved it is advisable, in order to prevent the spread of the disease, to treat the whole region actively when it may be done without any bad effect; or the application should be rubbed into the entire scalp, and the plaster-like application made to diseased areas only. In these hospital cases, however, I have treated diseased and non-invaded parts alike, excepting, of course, in the matter of depilation. Under the various plans of treatment the disease after several weeks begins to show signs of lessened activity, and in two or three months more it seems, in some cases, apparently cured, no evidence, microscopic or visual, appearing during an observation of one or two weeks after treatment is suspended. As already remarked, however, this apparent cure is rarely complete in this short a time, and a recrudescence, less extensive usually, is seen to follow one or two months after remedial applications have been discontinued. It is only by persistence that a final cure, permanent in character, is to be looked for; and unfortunately the patient's hearty co-operation is so necessary for this, and usually after a short period this is lacking or so imperfectly given, that it is but seldom that one can honestly say that he has permanently cured a case of this disease. Indeed, a cure is so exceptional for the various reasons given, and as the large majority of these cases are imported patients, and usually of the lowest scale of humanity, that in my judgment the admission on our shores of emigrants, children or adults with this contagious disease should be forbidden. Further, it seems an unnecessary burden to place upon our city to permit parents who have the means of livelihood to place an affected child in the hospital for the treatment of this disease, and the same holds true as to the admission of adults who are in nowise incapacitated; with a half to one hour's daily attention at home cases can be just as well cared for under dispensary supervision.

CIRCUMSCRIBED SCLERODERMA, WITH REPORT OF A CASE.

By J. ABBOTT CANTRELL, M.D.

Morphœa (μορφή), or circumscribed scleroderma, is very generally admitted at the present day to be a variety of scleroderma occurring in a circumscribed form, although certain writers are inclined to consider the disease as entirely distinct from scleroderma.

A great many different names have been given to the affection at various times, thus giving rise to much confusion. Some observers regard morphœa as allied to true leprosy, and use the term synonymously with elephantiasis græcorum, which is a very unfortunate application of the name. Hilton Fagge[1] thought there existed a strong resemblance between morphœa and scleroderma, and according to the researches of Crocker[2] it would appear there is a relationship between the affections.

The disease is usually described as being characterized by the appearance of one or more patches, lines or bands which are smooth or slightly scaly, varying in size, oval, elongate or roundish in shape, sometimes somewhat elevated or on a level with the neighboring skin, and conveying a sensation of firmness to the touch. The lesions are surrounded by a lilac-colored tint, showing dilated capillaries. Usually they are well defined and present a smooth ivory-like surface, sometimes shining and atrophic in appearance. The lesion has been likened to a piece of bacon or ivory deposited in the cutis. The color varies from pink to yellow, brown, and at times black. The patches are, as a rule, unilateral and may follow the course of a superficial nerve or nerves as witnessed in herpes zoster. When atrophy has progressed in the patch, owing to the infiltrated condition of the affected part, it is generally a matter of difficulty to pick up the tissue between the fingers. When the patch has fully developed it

[1] Guy's Hospital Reports for 1868.
[2] Path. Trans., vol. xxxi. p. 315.

shows a tendency either to become contracted or to a complete return to the normal condition of the skin.

The name morphœa has been given to the lesions when they are quite white in appearance, and that condition in which much pigmentation exists has been designated as morphœa nigra. The variety in which the patches appear dry and resemble parchment is known as morphœa atrophica.

The lesions may appear on different parts of the body; they show, however, a predilection for the face, neck, back, abdomen, arms, thighs and mammæ. They do not show any tendency towards symmetry. The secretion of sweat may be diminished or entirely suppressed in proportion to the existing atrophy of the affected part. Usually the symptoms consist only of slight pruritus or suppression of sweating in the affected area; at times pain and tingling may be experienced by the patient; this is, however, exceptionally the case. Sensibility to tactile and painful impressions is rarely impaired. As a rule, the course of the disease is gradual, frequently requiring months until it has reached its full development. In exceptional cases, after the disease has existed for months or years, it may cause marked atrophy and decided loss of mobility and deformity. An ulcerated condition of the patches has also been reported.

Much obscurity prevails as to the etiology of the disease. It appears to be due to a disordered condition of the nervous system. It is by no means a frequent affection, and, according to the statistics of the American Dermatological Association, there appeared but one case in 18,683 consecutive cases of cutaneous diseases. It is oftener met with in females than males, and may occur at any age, although very young children (after the second year) and young adults are more prone to be affected. Traumatic influences also seem to act as an exciting cause, as in the case reported by Hilton Fagge of a woman in whom lesions appeared where she wore garters. Hutchinson[1] describes a case in which a white patch was noticed in front of the instep, from where it began to spread, supposed to be due to the rubbing of the boot. Crocker[2] refers to several cases, including a few in which the patches seem to have followed injury or friction of the clothing. W. Allen Jamieson[3] reports a case in which alopecia was

[1] Clinical Lectures, vol. i. p. 315.
[2] London Lancet 1886, vol. iv.
Archives of Dermatology, N. Y., 1881, p. 141.

also witnessed, the morphœa not affecting the hairy parts of the body. The author's reason for reporting this case lies in the fact of but few cases being due to traumatism having been recorded, and whereas, as has already been remarked, the etiology of morphœa is usually obscure.

It appears in the case that I am about to describe to be entirely due to traumatic influences.

N. S., a fairly well developed and nourished girl of eight years, was brought to me on the 7th of July, 1893, for an affection occupying the right side of the head and forehead.

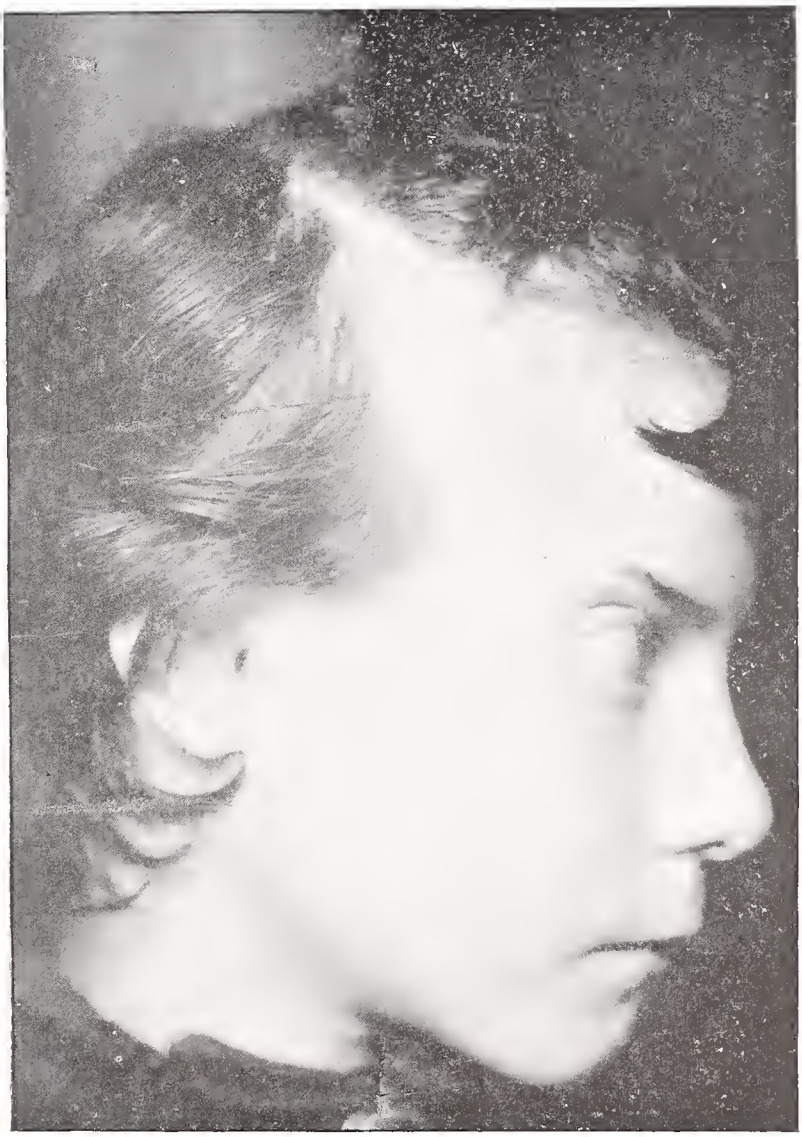
Her mother, who accompanied her, was an ordinary-sized American woman, who had always been in perfect health. The father was a well-developed Irishman. There were three other children in the family, the patient being the oldest, and all were said to be in good health.

The patient states that about eighteen months before her visit to me, while romping at school, she fell and struck her head upon the pointed edge of a school desk. This was followed by the usual inflammation and swelling; the condition subsiding in a few days, and nothing more was thought of the occurence until three months afterward (fifteen months before these notes were taken), when visiting her aunt, it was noticed that the hair was falling from the patch. As the hair continued to fall, her aunt became frightened and consulted a physician, who placed the child under treatment, but the child, getting no better, came under my care on the first-mentioned date.

The diseased area was made up entirely of one patch, and was situated upon the right side of the temporal region and forehead, being six or seven inches in length and ranging from one inch at its upper to two or more at its lower border. The part bears much resemblance to an ordinary burn-cicatrix, but on close inspection no evidence of such a condition is present. In color and appearance it resembles an ordinary billiard ball, being shiny, hard and ivory-like, and is entirely devoid of hair, with the exception of possibly some few straggling hairs near its border. The patch begins at a point of the parietal suture, on a line with the parietal boz (the point upon which the bruise occurred), and as it extends downward toward the eye it broadens out, thus resembling a triangle in shape. Besides being ivory-like and shiny, it is smooth and perfectly white, showing a decided contrast with the surrounding skin. It is nowhere œdematous, does not pit upon pressure, nor can the patch be pinched up between the fingers; it seems to be directly adherent to the bone, and there exists apparently no subcutaneous tissue at this point. The entire diseased area is drawn and tightened considerably, and while this condition is apparent at its upper border, it is still more so at its lower border. due to the external canthus of the eye being drawn up. The patch differs in many respects from others on account of having linear streaks through it and not around its borders; it seems to be normal in feeling, the patient being readily able to distinguish between hot and cold applications; she feels the point of a pin and can tell the difference between it and the head.

She complains of no unnatural sensation in the part; there is no ulceration present. Climatic changes from cold to hot or hot to cold do not affect the diseased area at all. At the time of my first seeing her the condition seemed to be at

FIG. 1.



Circumscribed scleroderma.

a standstill. She was placed on a placebo, and at my subsequent visit galvanism was applied for fifteen minutes daily (about ten cells of a Fleming constant galvanic battery).

In describing the pathology of this curious affection Crocker uses the following words: "It appears that, owing probably to some defect in innervation, cell-exudation occurs round the vessels, narrowing the lunula, obstructing, therefore, the blood-flow and leading to thrombosis, and sometimes to a real rupture and effusion. Each atrophic spot seen near a growing patch is the base of a cone from which the blood-supply is cut off, the violet zone being due to the collateral hyperæmia round an anæmic area. The patch or atrophic spot thickens by the fibrillation of the effused cells. Where the arterial supply is cut off, an atrophic spot only is produced; where it is only diminished, partial atrophy with connective-tissue hyperplasia or morphœa is developed."

The results obtained by treatment are not very encouraging, but attention must be paid to the general health. The administration of tonics, such as cod-liver oil, strychnia and arsenic, and a residence in a suitable climate are useful. Local treatment has failed to exert a beneficial effect on the disease. Brocq reports good results obtained by the use of electrolysis in two cases. It is thought that galvanism, applied locally, may be of some benefit. Massage has also had its followers.

A TABULAR REPORT OF TWO HUNDRED AND FIFTY-FOUR AUTOPSIES.

By W. B. JAMESON, M.D.

The Philadelphia Hospital, unlike many of the great hospitals, does not permit an autopsy on the body of every person who dies in the institution. The preliminaries to an examination of a dead body are as follows: A written permission from the relatives or friends of the deceased is the first consideration, without it an autopsy is not possible. A document signed by the visiting physician, the chief resident physician and the superintendent provides the pathologist with the necessary authority to proceed. In case the body is not claimed by a relative, friend or association, the latter document only is required. On account of unavoidable delay in obtaining the requisite permission, few autopsies are made earlier than twenty-four hours after death, and it is not at all uncommon for the examination to be delayed to thirty-six or forty-eight hours.

When Prof. John Guitéras became the pathologist of the hospital he devised a scheme for the examination of the body and the autopsy record which is shown in the sample sheet. This is an excellent order, and has been followed in making these examinations. By a reference to the sample sheet of the post-mortem record-book it will be observed that the name, age, sex, color, etc., are printed at the top of the left page. A space is left for the clinical diagnosis and the clinician's name, also a space for the pathological diagnosis and the pathologist's name. The sequence of examination is also printed. The remainder of this page and the right page are devoted to the notes of the autopsy.

Since June, 1892, there have been two hundred and fifty-four antopsies performed in the mortuary of the hospital by Dr. Guitéras and myself. Six of these were in babies unable to assimilate sufficient food to maintain life, but did not reveal any gross lesion. The others

all had a more or less abundant pathological diagnosis, which constitutes the following summary:

Abdominal section, recent	1
Abscess of ankle	2
of brain	1
of broad ligament and ovary	1
of hand	1
of heart	1
of liver	3
of lung	6
of perineum	1
of prostate	1
of scalp	1
of spleen	1
of wrist	1
Adenoma of ovary	1
Anæmia, pernicious	2
Angioma of face	1
Aneurism of arch of aorta	7
in pulmonary tubercular cavity	2
in splenic artery	1
in thoracic aorta	1
Aorta, abdominal, rupture of	1
Aortic ring, fibroid thickening of	1
Appendicitis, acute	1
gangrenous with preforation	1
Arterio-fibrosis	26
Artificial anus, recent	1
Arteritis with partial occlusion of right cerebral artery	1
with partial occlusion of left cerebral artery	1
with total occlusion of right cerebral artery	1
Atheroma of aorta	23
of cerebral arteries	2
of coronary arteries	2
Biliary ducts, obstruction of common by stone	1
obstruction of cystic by stone	1
Bladder, hypertrophy of walls	4
Bronchiactasis	2
Bronchitis, acute	3
chronic	2
gangrenous	1
ulcerative	1
Bubo	1
Calculus pyelitis	4
Calvarium, great thickening of	6
Carcinoma of bladder	1
of brain	1
of breast (primary)	2
of bronchial glands	1
of frontal bone	1
of liver	5
of lungs	2
of peritoneum	3
of pleura	1
of retro-peritoneal glands	2
of spleen	1
of stomach (primary)	3
of suprarenal body	1
of uterus	1
Cerebral hemorrhage	3
softening	5
Cerebro-spinal fluid, excess of	11
Chancroid, phagodenic	1
Colitis, follicular	2
Cut throat	1
Cyanotic induration of kidney	9
of liver	6
of lungs	3
of spleen	3
Cyst, hemorrhagic of Fallopian tube	1
from hemorrhage in internal capsule	4
from hemorrhage in external capsule	1
from hemorrhage in lenticular nucleus	2
from softening in caudate nucleus	2
from softening in temporal lobe	1
from softening in internal capsule	2
from softening in lenticular nucleus	5
from softening in optic thalamus	1
from softening in pons	3
of liver	1
of ovary	1

Cystitis 11
diphtheritic 2
hemorrhagic 1
Duodenum, cicatricial contraction of 1
congenital occlusion of 1
healed ulcer of 1
ulcer of 1
Dysentery, acute 7
diphtheritic 6
chronic 8
Embolism of right renal artery 1
Emphysema, interstitial, around trachea and in anterior and superior mediastina 1
vesicular 26
Empyema, pleural 4
anterior ethmoidal cells 1
frontal sinus 1
Endocarditis, ulcerative 3
vegetative, acute, mitral 5
aortic 4
Endometritis, diphtheritic, of placental site 1
of cervix 1
Enteritis, catarrhal 1
follicular 3
diphtheritic 1
Entero-colitis 2
Epithelioma of bladder 1
of ovary 1
of uterine cervix 1
Erysipelas of external genitals 1
Fracture of ribs 2
Fibrous deposit on floor of skull 2
Fibroma of kidney 1
of liver 1
of stomach 1
of suprarenal body 1
Gastritis, chronic 1
Gastro-enteritis 1
Gall-bladder, obliteration of 1
Gall-stones 9
Gumma of liver 2
of pancreas 1
Hemorrhage in chest-wall 1
in intestine 1
in mucous membrane of intestine 1
Hemorrhage in mucous membrane of stomach 1
in œsophagus 1
in peritoneal cavity 7
in pleural cavity 3
in pons 1
in stomach 1
in spinal canal 1
Hæmatoma, dura 5
subperitoneal 1
of scalp 1
Hemorrhages, multiple, under pericardium 1
under peritoneum 1
under pleura 3
gastro-intestnal 1
subdural 1
subpial 1
Hernia, abdominal 1
strangulated inguinal 1
Heart, brown atrophy of 1
fatty infiltration of 6
fatty degeneration of 7
patulous foramen ovale of 1
Hydrothorax 17
Hypertrophy of left ventricle of heart 39
of right ventricle of heart 11
Infarct, hemorrhagic, of left auricular appendix 1
of right ventricular wall 1
Intestines, amyloid degeneration 2
diverticulum 2
perforation of 3
typhoid ulcers of 9
Jaundice, obstructive 4
Kidney, amyloid degeneration 7
cystic 9
dilated pelvis 3
displaced 1
embolic abscess 1
hemorrhagic infarct of 3
horseshoe 2
laceration 1
miliary abscesses 2
penetrating wound 1
Laryngitis, syphilitic 1
Leukæmia 1
Lipoma of colon 1

Liver, amyloid degeneration..... 7
cavernous angioma..... 2
cirrhotic..... 19
echinococcus cyst..... 1
fatty degeneration..... 3
fatty infiltration..... 27
hemorrhage into parenchyma.. 1
Lungs, anthracosis..... 6
collapse..... 7
fibroid induration..... 2
gangrene..... 5
hypostatic congestion.. 4
œdema..... 10
purulent lymphangitis..... 1
Luxation of neck..... 1
Meningitis, chronic cervical..... 2
chronic lepto-..... 1
Mesentery, hemorrhagic infiltration of..... 1
Myelitis, transverse with softening 2
Nephritis, acute parenchymatous... 16
acute suppuration..... 2
chronic interstitial..... 82
chronic parenchymatous..... 29
hemorrhagic..... 2
Osteoma of falx cerebri..... 4
on border of foramen magnum 1
Pachymeningitis, purulent..... 1
Parotitis, purulent..... 1
Pancreas, amyloid degeneration.... 1
scirrhus carcinoma..... 1
Peribronchitis..... 3
Pericarditis, acute plastic..... 7
acute purulent..... 1
chronic fibrinous..... 4
hemorrhagic..... 1
Pericardial effusion..... 12
Pericardium, calcareous infiltration 1
Pericncephalitis with atrophy of cortex..... 2
Perisplenitis..... 6
Peritonitis, acute general..... 13
acute local..... 1
chronic general..... 3
hemorrhagic..... 1
Peritoneal effusion..... 8
Peritoneum, encephaloid carcinoma..... 1
scirrhus carcinoma..... 1
Phlegmasia alba dolens..... 1
Phthisis, fibroid..... 1
Pleurisy, acute plastic..... 34
chronic fibrous..... 37
hemorrhagic..... 1
Pleural effusion..... 18
hemorrhagic..... 2
Pneumonia, catarrhal..... 19
chronic..... 4
croupous..... 47
fibroid..... 2
inspiration..... 6
Prostate, adeno-carcinoma..... 1
hypertrophy..... 3
Purpura hemorrhagica..... 1
Pyelitis..... 5
hemorrhagic..... 2
Pyo-pneumo-thorax..... 1
Pyosalpinx..... 1
Pyothorax..... 2
Rupture of mesenteric artery..... 1
Rachitis..... 1
Sarcoma of corpus callosum..... 1
of liver..... 2
of lung..... 1
of pancreas..... 1
of retro-peritoneal glands..... 2
of suprarenal body..... 1
of superior mediastinum..... 1
of thigh and pelvis..... 1
Sclerosis of brain and cord, disseminated..... 1
of temporal lobe..... 1
Spleen, amyloid..... 6
infarct..... 6
Splenic tumor..... 12
Stomach, amyloid..... 1
atrophy of mucous membrane. 2
biventral..... 1
encephaloid..... 2
rupture..... 1
ulcer..... 1
Suprarenal body, laceration of..... 1
Synovitis, purulent, of foot..... 1
of knee..... 2
Syringo myclia..... 1
Thrombosis of right iliac artery..... 1
of veins of leg..... 1
Tonsilitis, ulcerative..... 1

Tracheotomy	1
Trachitis, gangrenous	1
Tuberculosis of bladder	1
of bodies of vertebræ	2
of duodenum	1
of intestine, large	10
of intestine, small	20
of kidney	17
of larynx	1
of liver	7
of lung	53
of lymphatic glands	13
of meninges of brain	4
of meninges of cord	1
of pericardium	2
of peritoneum	3
of pleura	11
of spleen	5
of stomach	2
Tuberculosis of suprarenal body	5
of thymus	1
of trachea	1
of ureter	1
of vermiform appendix	3
Tyroma	1
Ureteritis, hemorrhagic	1
ulcerative	2
Ureter, dilated	3
obstructed	1
Urinary infiltration of external genitals	2
Ulcer of leg	3
of œsophagus	1
Uterus, myofibroma	7
procidentia	1
Valvulitis, chronic, sclerotic, aortic	19
mitral	31

The oldest persons whose autopsy record appears in this series reached the age of eighty-eight. One was a colored man and the other was a white woman. At the other extreme there are still-births. The following table shows the sex and mortality per decade:

Decade.	First.	Second.	Third.	Fourth.	Fifth.	Sixth.	Seventh.	Eighth.	Ninth.
Male	13	1	20	25	36	36	20	10	8
Female	8	2	11	15	10	18	12	8	1

VOLUME VIII., 189.....

PHILADELPHIA HOSPITAL—POST-MORTEM RECORD, No.....

189

Name, Age, Sex, Color, Nationalty,
Ward, Bed, Admitted, Attending Physician,
Autopsy ordered by Date and hour of death, Date and hour of autopsy,

Clinical Diagnosis.

Pathological Diagnosis.

Signature of Resident Physician,

Pathologist.

Notes of the Autopsy.—The Pathologist will observe the following order of record, when practicable:

I. External appearances,
II. General description of Abdominal Cavity,
III. Thoracic Cavity and Pleural Sacs,
IV. Pericardium,
V. Heart,
VI. Left Lung,
VII. Right Lung,
VIII. Spleen,
IX. Left Suprarenal Capsule,
X. Left Kidney,
XI. Right Suprarenal Capsule,
XII. Right Kidney,
XIII. Ureters and Bladder,
XIV. Rectum,
XV Internal Genital Organs,
XVI. External Genital Organs,
XVII. Duodenum,
XVIII. Stomach,
XIX. Pancreas,
XX. Liver,
XXI. Mouth and Neck. Œsophagus. Trachea.
XXII. Large Arteries and Veins,
XXIII. Semilunar Ganglion,
XXIV. Thoracic Duct,
XXV. Brain and Spinal Cord,
XXVI. Intestines,
XXVII. Extremities,
XXVIII. Microscopic Examination,

LIST OF THE FORMER RESIDENT PHYSICIANS OF THE PHILADELPHIA HOSPITAL.[1]

By EDWARD R. STONE, M.D., and W. A. N. DORLAND, M.D.

ABBOTT, HARVEY N.	1523 Fontaine St., Phila.	79	U. of P.
ABBOTT, W. L.		84	U. of P.
ABEL, FRED. T., dec'd.		76	U. of P.
ALBERTSON, WILLIAM C.	Belvidere, N. J.	86	U. of P.
ALEXANDER, CLARA J.	Johnstown, Pa.	89	W. M. C.
ALLEN, HARRISON.	1933 Chestnut St., Phila.	61	U. of P.
ALLIS, OSCAR H.	1604 Spruce St., Phila.	66	Jeff.
ALLISON, E. W.	125 S. 18th St., Phila.	80	U. of P.
ALLYN, HERMAN B.	310 N. 40th St., Phila.	85	U. of P.
AMES, ROBERT P.	Springfield, Mass.	81	Jeff.
ANDERSON, GEORGE B.	Latrobe, Pa.	77	Jeff.
ASHTON, THOMAS G.	1533 Pine St., Phila.	88	Jeff.
ATCHISON, WILLIAM A.	15 S. Cherry St., Nashville, Tenn.	52	
ATLEE, JR., WILLIAM A.		87	U. of P.
AUGÉ, TRUMAN	2802 N. Broad St., Phila	92	
ANGNEY, WILLIAM M.	519 Spruce St., Phila.	79	Jeff.
BAKER, P. B. L.	Enfanta, Barbour Co., Ala.	49	U. of P.
BALDY, J. M.	1722 Chestnut St., Phila.	84	U. of P.
BALL, EDWARD S., dec'd.	Zanesville, Ohio	78	

[1] The following list has been prepared by Drs. Stone, Dorland and C. K. Mills, and has been received with the letter which follows. Any corrections or additions will be gladly received by Dr. Edward R. Stone or by the Editor of these REPORTS.

PHILADELPHIA, May 20, 1895.

DEAR DOCTOR DE SCHWEINITZ:

I send herewith the list of Blockley ex-Residents. The number in the third column refers to the year of appointment to the hospital; the fourth column, to the college.

U. of P., University of Pennsylvania; Jeff., Jefferson Medical College; W. M. C., Woman's Medical College of Pennsylvania; Medico-Chi., Medico-Chirurgical College; Univ. City N. Y., University of City of New York; Penna. M. C., Pennsylvania Medical College (extinct).

I should be glad to have the list more complete, and if you will incorporate a request to that effect, you would oblige,

Yours sincerely,

EDWARD R. STONE.

The list is that of the "Association of ex-Resident and Resident Physicians of the Philadelphia Hospital."

Barksdale, R.	Petersburg, Va.	52	U. of P.
Bartles, William H.	Flemington, N. J.	65	Jeff.
Barrister		43	
Beecher, A. C. W., dec'd.	Philadelphia.	67	Jeff.
Bellows, Horace M.	Huntingdon Valley, Pa.	61	U. of P.
Benton, Charles H., dec'd		59	
Benton, John H., dec'd.	Ogdensburg, N. Y.	48	U. of P.
Berens, Bernard.	2002 Chestnut St., Phila.	80	U. of P.
Berens, Joseph, dec'd.	New York.	75	
Berens, T. Passmore.		87	U. of P.
Bertolette, D. N.	U. S. N.	72	Jeff.
Beyea, Harry D.	237 S. 13th St., Phila.	91	U. of P.
Birkey, Thomas W.	245 S. 6th St., Phila.	51	U. of P.
Bitting, Miriam		89	
Black, John J.	New Castle, Del.	65	U. of P.
Blackford, Benjamin	West'n Lunatic Asylum, Staunton, Va.	55	Jeff.
Bliss, A. A.	1832 Race St., Phila.	83	U. of P.
Bloomfield, J. C.	Athens, Ga.	88	Jeff.
Boardman, Charles H.	16 Wesley Ave., Evanston, Ill.	62	U. of P.
Boenning, H. C.	538 N. 6th St., Phila.	79	Jeff.
Botsford, William, dec'd.		68	Jeff.
Bower, J. L.	Reading, Pa.	88	Jeff.
Boyd, John S.	19 S. 12th St., Phila.	70	U. of P.
Boyer, Z. P.	1829 N. Broad St., Phila.	81	U. of P.
Bradfield, G. M.	Philadelphia Hospital.	91	Jeff.
Bradley, Alfred E.	U. S. A.	87	Jeff.
Bradley, Michael.		58	
Brady, Elliot T.	1600 Wesley Ave., Evanston, Ill.	86	Jeff.
Bragg, J. C.		58	
Braxton, Tomlin.	Palls, King William Co., Va.	54	U. of P.
Brechemin, L.	U. S. A.	76	U. of P.
Bricker, Charles E.	2739 Girard Ave., Phila.	81	Jeff.
Brooke, Benjamin.	U. S. A.	89	U. of P.
Brooke, Harriet W.	Reading, Pa.	86	W. M. C.
Brown, C. H.	1824 Diamond St., Phila.	78	U. of P.
Brown, G. S.	114½ 21st St., Birmingham, Ala.	85	Jeff.
Bruen, E. T., dec'd.	Philadelphia.	74	U. of P.
Bruner, W. E.	Cleveland, Ohio.	91	U. of P.
Buck, Samuel T.		90	
Buck, W. P.	133 N. 19th St., Phila.	69	U. of P.
Budd, A. V.	Lockville, Chatham Co., N. C.	54	U. of P.
Bunce, T. S.		82	U. of P.
Burtenshaw, J. H.	284 Amsterdam Ave., N. Y.	91	U. of P.
Burnett, J. W., dec'd.		62	Jeff.
Burwell, Geo. N., dec'd.	Buffalo, N. Y.	43	
Bush, Lewis P., dec'd.	Wilmington, Del.	36	U. of P.
Campbell, H. E.	100 Sheffield St., Allegheny City, Pa.	81	Jeff.
Campbell, H. S.	828 N. Broad St., Phila.	67	U. of P.
Carpenter, W. H.	Salem, N. J.	92	U. of P.
Carter, Charles, dec'd.	Philadelphia.	40	U. of P.
Carter, W. S.	807 N. 41st St., Phila.	90	U. of P.
Chase, A. F.	3805 Baring St., Phila.	74	Jeff.
Chase, Robert H.	State Hospital, Norristown, Pa.	69	U. of P.
Christie, S. P.		58	
Coleman, J. S., dec'd.	Augusta, Ga.	57	Jeff.
Coleman, P. T.	Curdsville, Buckingham Co., Va.	52	
Cone, Claribel.		91	W. M. C.
Corbet, W. B., dec'd.		63	
Cousins, A. S.		58	U. of P.

Cowgill, Clayton A.	3609 Baring St., Phila.	46	U. of P.
Coyle, Robert.	723 S. 16th St., Phila.	85	Jeff.
Craig, William G.		92	
Craig, T. C.	2002 Chestnut St., Phila.	80	U. of P.
Crowell, Elisha.	21 S. 39th St., Phila.	51	U. of P.
Crowell, G. M.		82	
Cullen, J. S. Dorsey.	412 E. Grace St., Richmond, Va.	54	
Culpepper, W. A., dec'd.		83	U. of P.
Cummings, J. B.	Forrest City, Ark.	73	Jeff.
Cummiskey, James.	2026 Vine St., Phila.	56	Jeff.
Curry, G. E.	Wills Eye Hospital, Phila.	92	
Curtin, Roland G.	22 S. 18th St., Phila.	66	U. of P.
Curtis, L. W.	U. S. N.	80	U. of P.
Da Costa, J. Chalmers.	2050 Locust St., Phila.	85	Jeff.
Daggett, William G.	22 College St., New Haven, Conn.	84	U. of P.
Darby, J. F., dec'd.		58	U. of P.
Darrach, George M.	Cumberland, Monroe Co., Ind.	52	
Dashiel, T. K.		58	
Davis, A. M.	5054 Main St., Germantown, Pa.	92	U. of P.
Davis, James A.	527 S 42d St., Phila.	87	U. of P.
Davis, Sidney.	Milton, Pa.	78	U. of P.
Davis, T. D.	6018 Penn Ave., Pittsburgh, Pa.	70	Jeff.
Day, George E.	Strasburg, Pa.	84	Jeff.
Dease, Stephen S.	India	75	Jeff.
Deaver, R. W.	6033 Main St., Germantown, Pa.	74	U. of P.
De Benneville, J. S.		59	U. of P.
Dercum, Clara T.	1115 Fairmount Ave., Phila.	87	W. M. C.
Dessau, S. Henry.	47 W. 56th St., N. Y.	68	Jeff.
Dillon, J. D.	328 S. 5th St., Phila.	79	Jeff.
Diller, Theo.	Westinghouse Building, Pittsburgh, Pa.	86	U. of P.
Dinsmore, F. M.	E. Washington, N. H.	94	U. of P.
Doan, Henry H.		90	
Dock, C.	904 N. Broad St., Phila.	80	Jeff.
Dodge, C. L.		78	
Dolan, W. K.	Scranton, Pa.	80	U. of P.
Donnelly, M. A., dec'd.		79	Jeff.
Donohue, Michael J.	Waterbury, Conn.	86	U. of P.
Dorland, W. A. N.	120 S. 17th St., Phila.	86	U. of P.
Dripps, J. H.	1812 N. 11th St., Phila.	79	Jeff.
Duer, Edward L.	1606 Locust St., Phila.	60	U. of P.
Duhring, Louis A.	1411 Spruce St., Phila.	67	U. of P.
Dulles, Charles W.	4101 Walnut St., Phila.	75	U. of P.
Dwight, Henry E.	336 S. 15th St., Phila.	67	U. of P.
Eberman, Henry F.	Lancaster, Pa.	79	U. of P.
Edgar, John M.	U. S. N.	80	U. of P.
Edwards, Joseph S.	Atlantic City, N. J.	75	U. of P.
Edwards, W. A.	San Diego, Cal.	81	U. of P.
Edwards, W. F.	Detroit, Mich.	80	U. of P.
Elder, F. H.	1427 Arch St., Phila.	82	U. of P.
Elmer, Henry W.	65 W. Commerce St., Bridgeton, N. J.	69	U. of P.
Elmer, William, dec'd.	Bridgeton, N. J.	36	U. of P.
Eshleman, E. E., dec'd.	Philadelphia	70	Jeff.
Eshner, A. A.	224 S. 16 St., Phila.	88	Jeff.
Esterly, D. E.		94	U. of P.
Evans, E. W.	65 N. 3d St., Easton Pa.	84	U. of P.
Eversfield, W. C.	Agricultural College, Prince George's Co., Md.	63	U. of P.

Fairfield, J. H.	Great Falls, Mon.	84	U. of P.
Farnham, Alice May	Asylum, Hart's Island, N. Y.	84	W. M. C.
Fell, Jonathan		40	U. of P.
Flick, L. F.	736 Pine St., Phila.	79	Jeff.
Ford, William H.	1622 Summer St., Phila.	64	Jeff.
Fox, George Henry	18 E. 31st St., N. Y.	69	U. of P.
Frase.		82	
Free, G. B. M.	Williamson School, Pa.	84	U. of P.
French, Samuel	24 N. Franklin St., Wilkesbarre, Pa.	94	
Freund, H. H.	1310 S 5th St., Phila.	80	Jeff.
Garrett, E. F., dec'd	Philadelphia	77	Jeff.
Gaylord, H. R.		94	
Gibb, Joseph S.	817 Franklin St., Phila.	80	U. of P.
Gilbert, John E., dec'd	Gettysburg, Pa.	78	U. of P.
Gilmore, A.		91	W. M. C.
Girvin, Edward R.	Denver, Col.	76	U. of P.
Girvin, Robert M.	3906 Walnut St., Phila.	62	Jeff.
Githens, W. H. H.	1512 Pine St., Phila.	66	U. of P.
Goodman, H. E.	1509 Walnut St., Phila.	59	U. of P.
Gorgas, S. R., dec'd	Harrisburg, Pa.	75	Jeff.
Gotwald, D. K.	York, Pa.	82	U. of P.
Graber, Leon J. K.	Harrisburg, Pa.	79	U. of P.
Graham, A. H.		58	U. of P.
Graham, E. E.	1713 Spruce St., Phila.	87	Jeff.
Grayson, Charles P.	233 S. 13th St., Phila.	82	U. of P.
Griffith, S. H.	U. S. N., Washington, D. C.	71	U. of P.
Gross, S. W., dec'd	Philadelphia	56	Jeff.
Guitéras, Daniel	329 W. 58th St., N. Y.	74	U. of P.
Guitéras, John	130 S. 39th St., Phila.	74	U. of P.
Guitéras, G. M.	U. S. Marine Service, 130 S. 39th St., Phila.	85	U. of P.
Guthrie, G. W.	47 S. Franklin St., Wilkesbarre, Pa.	73	U. of P.
Hackley, C. E.	144 W. 44th St., N. Y.	60	U. of P.
Haehnlen, W. F.	1616 Walnut St., Phila.	82	U. of P.
Hagy, J. A.	Dobbs Ferry, N. Y.	64	U. of P.
Haines, Josiah		44	Jeff.
Halberstadt, George H.	218 Market St., Pottsville, Pa.	78	U. of P.
Hale, George	4428 Paul St., Frankford, Phila.	70	U. of P.
Hall, John C., dec'd	Friend's Asylum, Frankford, Phila.	69	U. of P.
Hall, J. H.	U. S. N.	73	U. of P.
Hall, William R.		71	U. of P.
Hallowell, William H.	1338 1st Ave., Minneapolis, Minn.	80	U. of P.
Hamill, R. H.	344 S 16th St., Phila.	79	U. of P.
Hamill, Samuel M.	227 S. 20th St., Phila.	88	U. of P.
Hammond, Clara M.		87	W. M. C.
Hancock, E. C.		81	U. of P.
Hardy, Benjamin F., dec'd	San Francisco, Cal.	39	U. of P.
Harris, Charles M., dec'd	Philadelphia	70	U. of P.
Harris, T. J.	106 W. 79th St., N. Y.	89	U. of P.
Harrison, J. M.	Bryn Mawr, Pa.	79	U. of P.
Hawley, B. F.	417 N. 33d St., Phila.	82	Jeff.
Hazlett, E. E.	Abilene, Dickinson, Co., Kan.	80	U. of P.
Hearne, James C.	Hannibal, Mo.	72	Jeff.
Heath, William H.		78	Jeff.
Heller, Jacob B.	Easton, Pa.	78	U. of P.
Helm, William H.	Sing Sing, N. Y.	64	U. of P.
Henderson		77	
Henley, Leo	Williamsburg, Va.	47	U. of P.
Henry, C. P.		82	U. of P.
Hewitt, George A.	878 N. 25th St., Phila.	77	Jeff.

Hickman, H.	2619 Columbia Ave., Phila.	84	U. of P.
Higginbotham, Edw'd G.	239 W. Walnut, Louisville, Ky.	45	U. of P.
Hinkle, William H.	1300 Spring Garden St., Phila.	92	U. of P.
Hitschler, William.		94	
Hitz, Henry B.	11th and Mineral Ave., Milwaukee, Wis.	91	
Hoffman, Joseph E.	126 Diamond St., Phila.	83	U. of P.
Hoffman, W. A., dec'd.		68	U. of P.
Holland, Daniel J., dec'd	Atchison, Kan.	76	Jeff.
Holland, J. W.		71	
Holmes, E. W.	1626 Mt. Vernon St., Phila.	80	U. of P.
Hoonaman, G. H.		90	
Horwitz, L. N.		82	
Horwitz, Theo., dec'd.		76	Jeff.
Hough, J. Stockton.	Trenton, N. J.	68	U. of P.
Houstoun, James P. S., dec.	98 Harris St., Savannah, Ga.	69	U. of P.
Housekeeper, F. P.	3508 Baring St., Phila.	74	U. of P.
Hug, Edward V	Navarro, Ohio.	93	
Hughes, D. E.	Philadelphia Hospital, Phila	90	Jeff.
Hughes, F. W.		80	U. of P.
Hull, George S.	72 W. Main St., Chambersburg, Pa.	77	U. of P.
Humphrey, G. E.	Hazleton, Pa.	90	U. of P.
Hunt, Elizabeth G.	1602 Arch St., Phila.	89	W. M. C.
Hurlock, F. J.	2161 N. 29th St., Phila.	82	Jeff.
Hutchinson, G. H.	Englishtown, N. J.	80	U. of P.
Hutchinson, Randall.	Eckman, W. Va.	87	U. of P.
Hutchinson, R. C.	817 Broad St., Trenton, N. J.	80	U. of P.
Irish, W. B.		94	
Jamar, John H.	Elkton, Md.	61	U. of P.
Jameson, E. W.		70	U. of P.
Jameson, William B.	761 N. 40th St., Phila.	86	U. of P.
Jamison, J. Ross.		94	
Janney, Frances S.	Riverton, N. J.	90	W. M. C.
Jenkins, S. R.	Prince Edward's Island.	84	U. of P.
Jenks, William F., dec'd.	Philadelphia.	66	U. of P.
Jessof, S. A. S.	Kittanning, Pa.	80	Jeff.
Jiminez, J. M.	Costa Rica.	69	Jeff.
Jiminez. S. M.	73 W. 36th St., N. Y.	79	Jeff.
Johnston, A. R.	Pierceton, Indiana.	82	Jeff.
Johnson, N. L.	Williamsport, Pa.	91	U. of P.
Kahn, Joseph.	St. Paul, Minn.	89	U. of P.
Keating, John M., dec'd.	Philadelphia.	74	U. of P.
Keating, Wm. V., dec'd.	Philadelphia.	44	U. of P.
Keefer, F. R.	U. S. A	89	U. of P.
Keller, Harry M.	State Miner's Hospital, Hazleton, Pa.	87	U. of P.
Kelly, E. P. B.		58	Jeff.
Kerlin, E. J.	576 Fullerton Ave., Chicago. Ill.	86	U. of P.
Kerr, J. W., dec'd.	Allegheny City, Pa.	77	Jeff.
Kerr, James W.	York, Pa.	39	U. of P.
Ketcham, S. R	1708 Green St., Phila.	89	U. of P.
King.		82	
King, William H.		94	
Kirk, L. H.		78	U. of P.
Kleinstuber, William S.	212 E. 6th St., Wilmington, Del.	93	
Koerper, Joseph.	471 N. 6th St., Phila.	62	Phila. Coll.
Knox, John.	Princeton, Iowa.	77	U. of P.
Kollock, Charles W.	Charleston, S. C.	81	U. of P.
Kugler, G. W.	North Carolina.	85	

LAKE, DAVID H.	Kingston, Pa.	85	Jeff.
LANDIS, H. G., dec'd.		70	Jeff.
LAPIN, F. S.		81	
LAZARUS, S. D., dec'd.	Philadelphia	83	Jeff.
LEE, CHAS. CARROLL, dec'd	79 Madison Ave., N. Y.	60	U. of P.
LEETE, JAMES M.	2912 Washington Ave., St. Louis	61	U. of P.
LEIDY, PHILLIP, dec'd.	526 Marshall St., Phila.	59	U. of P.
LEVY, HENRY H.	1407 E. Broad St., Richmond, Va.	71	U. of P.
LEVS, J. L.	State Hospital, Hazleton, Pa.	91	
LICHTY, JOHN A.		93	
LINCOLN, CLARENCE W.	2510 Columbia Ave., Phila.	93	
LINEAWEAVER, J. K.	Columbia, Pa.	61	Jeff.
LINN, G. WILDS.		73	U. of P.
LIPPINCOTT, FRANKLIN.		40	U. of P.
LITTIG, LAWRENCE W.	Iowa City, Iowa	84	U. of P.
LITTLE, W. T.		94	
LODER, PERCIVAL E.	517 S 8th St., Phila.	76	Jeff.
LODGE, JOHN.		59	
LODGE, WILLIAM J.	Baltimore, Md.	59	
LONG, WILLIAM S.	Haddonfield, N. J.	79	U. of P.
LUCAS, EMMA J.		93	W. M. C.
LUDLOW, JOHN L., dec'd.	1931 Chestnut St., Phila	41	U. of P.
LUDLOW, R. G.		64	U. of P.
LYMAN, GEORGE H., dec'd.	Boston, Mass.	43	
MACCOY, A. W.	1417 Walnut St., Phila.	70	U. of P.
MACCRACKEN, G. Y.	612 N. 13th St., Phila.	77	U. of P.
MAGOFFIN, M. M.	Mercer, Pa.	62	U. of P.
MANN, CHARLES W., dec'd.		66	
MARCUS, HERMAN D.	2263 Frankford Ave., Phila.	92	Medico-Chi.
MARTIN, CHARLES S.	11 S. 5th St., Allentown, Pa.	90	U. of P.
MARTIN, JOSEPH.	2009 Columbia Ave., Phila.	78	U. of P.
MATLACK, ELWOOD.	703½ N. 8th St., Phila	86	U. of P.
MATSON, E. G.	810 Penn Ave., Pittsburgh, Pa.	83	U. of P.
MATTHEWS, E. L. B.		91	W. M. C.
MATTHEWS, WILLIAM E.	181 Lincoln St., Johnstown, Pa.	87	Jeff.
MAURY, F. F.	Philadelphia	62	Jeff.
MCAULEY, JAMES A.	2827 Frankford Ave., Phila.	74	U. of P.
MCBRIDE, G. W., dec'd.		79	U. of P.
MCCAMY, R. H.	1932 E. Cumberland St., Phila.	79	
MCCARTY, R. H., dec'd.		79	U. of P.
MCCLEES, WILLIAM D.		85	U. of P.
MCCLINTOCK, JAMES, dec'd.		58	Jeff.
MCCOY, A.		88	Jeff.
MCCOY, HENRY W.	Golconda, Pope Co., Ill.	64	Jeff.
MCCONKEY, THOMAS G.	San Diego, Cal.	90	
MCCLURE, W. W.	21 S. 16th St., Phila.	64	Jeff.
MCDONALD		74	
MCFARLAND, JOSEPH.	1314 Franklin St., Phila.	89	U. of P.
MCGILL, GEORGE M., dec'd.	Princeton, N. J.	61	U. of P.
MCGOWN, D. J.	Los Angeles, Cal.	79	U. of P.
MCGUIGAN, J. J.	1604 Diamond St., Phila.	87	Jeff.
MCKEE, JAMES H.	1519 Poplar St., Phila.	92	U. of P.
MCKENYON		74	
MCLAUGHLIN, THOMAS N.	1814 N St., N. W., Washington, D. C.	82	
MCMILLAN, JAMES H., dec'd	St. Louis, Mo.	88	U. of P.
MCPHEETERS, WILLIAM M.	3452 Pine St., St. Louis, Mo.	40	U. of P.
MEANS, J. S.		59	
MEARS, J. EWING	1429 Walnut St., Phila.	66	Jeff.
MENAH, W. MCC.	1715 Pine St., Phila.	90	U. of P.
MERCUR, JOHN D.	1432 Pine St., Phila.	78	Jeff.

MERILLAT, WILLIAM C.		64	U. of P.
MILLIKEN, C. W.	Akron, Ohio	81	U. of P.
MILLIKEN, F. H.	3614 Walnut St., Phila.	79	U. of P.
MILLER, MILO G.	40 N. 36th St., Phila.	88	U. of P.
MILLER, ROBERT		64	Jeff.
MILLIGAN, JAMES E., dec'd.		72	U. of P.
MITCHELL, JAMES	711 N. 17th St., Phila.	83	U. of P.
MOFFITT, WILLIAM J.	1239 N. 2d St., Phila.	76	Jeff.
MONTEGUT, SIDNEY		92	
MONTGOMERY, E. E.	1715 Walnut St., Phila.	74	Jeff.
MOORE, DUNLAP		93	
MOORE, EDWARD M.	Rochester, N. Y.	39	U. of P.
MOORE, HENRY B.	Colorado Springs, Col.	86	Jeff.
MOORE, ISAAC H.	Prairie City, Iowa	78	U. of P.
MORRIS, S. E.		90	
MOSELY, E. B.	U. S. A.	68	
MOSS, WILLIAM	Chestnut Hill, Phila.	54	Jeff.
MOWRY, WILLIAM B.	212 North Ave., Allegheny City, Pa.	76	U. of P.
MOYER, SHERMAN T.	Shamokin, Pa.	86	U. of P.
MUHLENBERG, F., dec'd.	Lancaster, Pa.	68	U. of P.
MURRAY, G. D.	Scranton, Pa.	90	
MURRAY, JAMES M.		76	U. of P.
MURRAY, R. D.	U. S. A.	70	Jeff.
MUSSER, MILTON B., dec'd.	Philadelphia	68	Jeff.
MUSSER, JOHN H.	1927 Chestnut St., Phila.	77	U. of P.
MUTTART, GEORGE W.	505 Communipaw Ave, Jersey City.	91	Univ. City N. Y.
NEAD, D. W.	Harrisburg, Pa.	82	U. of P.
NEALE, H. M.	Upper Lehigh, Pa.	80	U. of P.
NEWGARDEN, G. J.	2024 Diamond St., Phila.	89	
NICHOL, WILLIAM L.	Nashville, Tenn.	49	U. of P.
NICHOLS, W. V.	Oceanside, Cal.	85	U. of P.
NICHOLSON, J. L.	465 Cooper St., Camden, N. J.	91	
NICKERSON, L. H. A.	Quincy, Ill.	75	U. of P.
NORRIS, RICHARD E.	Preston Retreat, Phila.	87	U. of P.
NOVAES, F. de P.	Brazil	84	U. of P.
OLIPHANT, N. B.	110 W. State St., Trenton, N. J.	80	U. of P.
OLIVER, CHARLES A.	1507 Locust St., Phila.	77	U. of P.
O'NEILL, J. WILKS	2110 Spruce St., Phila.	77	U. of P.
O'REILLY, THOMAS B.	2502 Richmond St., Phila.	94	
OWEN, J. J.	411 Pine St., Phila.	80	Jeff.
OWENS, JOHN E.	1806 Michigan Ave., Chicago	62	Jeff.
ORVIS, CHARLES, dec'd.		67	Jeff.
PARISH, WILLIAM H.	1435 Spruce St., Phila.	71	Jeff.
PARKE, WILLIAM E.	709 N. 17th St., Phila.	86	U. of P.
PARKHILL, CLAYTON	McPall Building, Denver, Col.	83	Jeff.
PARRY, JOHN S., dec'd.	Philadelphia	65	U. of P.
PATTERSON, JOHN P.		71	Jeff.
PECK, ELIZABETH L.	819 N. 40th St., Phila.	85	W. M. C.
PELHAM, J. W.		88	Jeff.
PERKINS, F. M	1428 Pine St., Phila.	76	U. of P.
PERRY, HEXT M.	South Carolina	73	Jeff.
PERRY, JOHN C.		40	U. of P.
PHILLIPS, WM. L., dec'd.	Pittsburgh, Pa.	76	U. of P.
PHILLIPS, R. J.	4011 Chestnut St., Phila.	83	Jeff.
PHILLRICK, INEZ C.		91	W. M. C.
PICOTT, MITCHELL H.	Geneva, N. Y.	61	Jeff.
PLUMER, A. J.	Whitman, Neb.	85	U. of P.

POLK, W. ROBESON	414 Nicollet Ave., Minneapolis, Minn	81	U. of P.
POLLOCK, FLORA		91	W. M. C.
POLTER, THOMAS C	5920 Green St., Germantown, Pa	71	U. of P.
PORTER, WILLIAM G	1118 Spruce St., Phila	69	U. of P.
PORTER, P. B	22 W. 31st St., N. Y	69	U. of P.
POTSDAMER, J. B	1333 Franklin St., Phila	80	Jeff.
POTTS, CHARLES S	1712 Wallace St., Phila	85	U. of P.
PREFONTAINE, L. A	13th St. and 2d Ave., N. Y	92	
PRESTON, SAMUEL P.		87	Jeff.
PRICE, HELENA J		86	W. M. C.
RABINOVITCH, LOUISA G		90	W. M. C.
RADEBAUGH, J. M	Pasadena, Cal	74	U. of P.
RANDALL, EDWARD, JR	Galveston, Texas	83	U. of P.
RANSLEY, ALEXANDER W	1230 S. 10th St., Phila	75	U. of P.
RATHBUN, F. D	New Windsor, Ill	79	Jeff.
REED, C. H	121 S. 17th St., Phila	82	U. of P.
REEDY, WALTER M		93	
REEVE, JOSIAH	Medford, N. J	64	U. of P.
RIESMAN, DAVID	2029 Wallace St., Phila	92	U. of P.
REX, OLIVER P	Penn M. Life Ins. Co., Phila	67	Jeff.
REYNOLDS, F. P	U. S. A	90	
RHEIN, JOHN H	318 S. 15th St., Phila	90	U. of P.
RICHARDSON, D. D	Norristown Insane Asylum	58	
RICHARDSON, ELLIOTT, dec.	Philadelphia	68	
RICHARDSON, GEORGE H	1724 Diamond St., Phila	91	U. of P.
RICHARDSON, JOHN D		39	U. of P.
RICIO, SEMFRIO, dec'd	Cuba	61	
ROBERTS, A. S	1901 Walnut St., Phila	77	U. of P.
ROBERTS, ISAAC E	1344 N. 13th St., Phila	67	U. of P.
ROBERTS, THOMAS S	22 S. 4th St., Minneapolis, Minn	85	U. of P.
ROBESON, W. F	Westinghouse Bldg, Pittsburgh, Pa.	85	U. of P.
ROBINSON, GEORGE S	House of Correction, Phila	81	U. of P.
ROBINSON, JOHN M		91	
ROHRER, GEORGE R	Lancaster, Pa	81	U. of P.
RONALDSON, WM. R., dec'd.	4017 Locust St., Phila	74	Jeff.
ROOKER, HERMAN S., dec'd		85	Jeff.
ROOT, M. P	India	83	W. M. C.
ROSA, W. V. V		39	U. of P.
ROSENAU, M. J	U. S. N	89	U. of P.
ROTHROCK, J. L		88	
ROUSSEL, A. E	2112 Pine St., Phila	82	Jeff.
RUSH, WILLIAM H	1822 Berks St., Phila	75	U. of P.
RYNIER, VAN NEST		80	U. of P.
SAILER, J	Philadelphia	92	
SCHIVELY, GEORGE S	1503 Gratz St , Phila	51	Jeff.
SCHNEIDEMAN, T. B	2725 N. 5th St., Phila	83	Jeff.
SEDGWICK, W. N		94	
SELTZER, C. JAY	1501 Walnut St., Phila	91	U. of P.
SEYMOUR, A. J	1209 Wallace St., Phila	86	U. of P.
SHARPLESS, CASPER W		88	U. of P.
SHARPLESS, WILLIAM T		88	U. of P.
SHEPPARD, J	36 Commerce St., Bridgeton, N. J	62	U. of P.
SHERARD, C. C., dec'd		60	U. of P.
SHERK, HENRY H	Pasadena, Cal	87	Jeff.
SHERMAN, M. H		91	W. M. C.
SHEW, A. M	Middleton, Conn	65	Jeff.
SHIMER, WILLIAM S	713 W. Cumberland St., Phila	86	U. of P.
SHIMMELL, JAMES S		81	Jeff.
SHUMWAY, E. A		94	U. of P.

SMALL, J. F.	135 E. Market St., York, Pa.	89	U. of P.
SMITH, A. S.	3226 Powelton Ave., Phila.	76	Jeff.
SMITH, ALLAN J.	Galveston, Texas.	86	U. of P.
SMITH, JR., CHARLES E.	St. Paul, Minn.	65	U. of P.
SMITH, H. A.	1523 Oxford St., Phila.	75	U. of P.
SMITH, ROBERT K., dec'd.		58	
SOMERS, GEORGE N. J.		94	
SOUTHERN, F. L.	628 N. 16th St., Phila.	90	
SPARKS, GEORGE W.	635 Spruce St., Phila.	65	Jeff.
SPENCE, J. E.		70	Jeff.
SPENCER, THOMAS R.		40	U. of P.
STAHL, B. F.	1502 Arch St., Phila.	87	U. of P.
STAMM, E. P., dec'd.		81	U. of P.
STEELE, J. DUTTON.	4504 Chester Ave., Phila.	93	U. of P.
STEHMAN, H. B.	Presbyterian Hospital, Chicago.	78	Jeff.
STENGEL, ALFRED.	332 S. 17th St., Phila.	89	U. of P.
STELWAGON, H. W.	223 S. 17th St., Phila.	75	U. of P.
STEVENS, A. A.	318 S. 15th St., Phila.	87	U. of P.
STEWART, A. H.	250 N. 12th St., Phila.	92	Jeff.
STEWART, W. H.	2620 N. 11th St., Phila.	82	U. of P.
STEWART, WALTER M., dec.		65	U. of P.
STILLÉ, ALFRED.	3900 Spruce St., Phila.	36	U. of P.
STIVERS, CHARLES G.	Tonawanda, N. Y.	92	
STONE, EDWARD R.	1701 Master St., Phila	72	Jeff.
STRYKER, S. S.	3833 Walnut St., Phila.	66	U. of P.
SUTTON, R. S.	419 Penn Ave., Pittsburgh, Pa.	65	U. of P.
TABB.		43	
TALLY, FRANK W.	1234 Spruce St., Phila.	87	U. of P.
TALLY, JAMES E.	55th and Lancaster Ave., Phila.	92	U. of P.
TAYLOR, G. B.	Barclay, Pa.	85	U. of P.
TAYLOR, J. L.		58	U. of P.
TAYLOR, ROBERT A.	Duluth, Minn.	86	Jeff.
TAYLOR, SARAH M.		90	W. M. C.
THOMAS, ADA R.		93	W. M. C.
THOMPSON, dec'd.	Pittsburgh, Pa.	78	
TOPPING, G. G.	Denver, Col.	73	Jeff.
TUCKER, HENRY.		94	
TUTEUR, EDWIN B.	LaCrosse, Wis.	90	
TUTTLE, JAMES P.	136 W. 41st St. N. Y.	81	U. of P.
UPSHUR, GEORGE L.		43	
VANDERVOORT, C. A.		94	
VAN GASKEN, F. C.	1132 Spruce St., Phila.	91	W. M. C.
VAN HARLINGEN, A.	118 S. 17th St., Phila.	68	U. of P.
VANNEMANN, W. S.	Persia.	89	U. of P.
VAN VALZAH, W. W.	10 E. 43d St., N. Y.	76	Jeff.
VOGLER, GEORGE W.	565 N. 5th St., Phila.	77	U. of P.
VOORHEES, N. W.	Danville, Pa.	83	U. of P.
VOORHEES, SHEPARD.		89	U. of P.
WALKER, JAMES B.	1617 Green St., Phila.	72	U. of P.
WALKER, JOHN S.		71	U. of P.
WALKER, THOMAS L., dec'd	Lynchburg, Va.	39	U. of P.
WALLACE, WILLIAM H., JR.		64	U. of P.
WALTERS, CHARLES, dec'd.		88	U. of P.
WAPLES, M. H.	Dubuque, Iowa.	65	Jeff.
WARE, J. D.	114 N. Delaware Ave., Phila.	76	U. of P.
WARING, JAMES J., dec'd.	Savannah, Ga	52	U. of P.
WEHNER, WILLIAM H.	119 Wistar St., Germantown, Phila.	87	Jeff.
WEIGHTMAN, JOHN F., dec.	Philadelphia.	66	U. of P.

WEIDMAN, W. M.	214 S. 5th St., Reading, Pa.	60	U. of P.
WELLS, GEORGE M.	Wayne, Pa.	85	U. of P.
WELLS, P. F.	4023 Brown St., Phila.	82	U. of P.
WELSH, JOHN C.		93	
WERKEL		77	
WESTON, GEORGE D.	98 Main St., Springfield, Mass.	87	U. of P.
WETHERILL, H. M.	1420 Chestnut St., Phila.	78	U. of P.
WHELAN		74	
WHITE, J. WILLIAM.	1810 S. Rittenhouse Square, Phila.	73	U. of P.
WHITEHEAD, P. F.		59	Jeff.
WILLARD, DE FORREST.	1601 Walnut St., Phila.	67	U. of P.
WILLETTS, E. MILES.	104 Adams St., Memphis, Tenn.	55	Jeff.
WILLITTS, C. H.	24 S. 18th St., Phila.	79	U. of P.
WILLS		82	
WILSON, F. G.		91	
WILSON, JAMES F., dec'd.	Philadelphia.	65	U. of P.
WILSON, JOHN J.		43	
WILSON, W. R.	267 S. 21st St., Phila.	87	Jeff.
WOLLERTON, S. H.	Oxmoor, Ala.	80	U. of P.
WOOD, B. S.		58	
WOOD, H. C.	1925 Chestnut St., Phila.	62	U. of P.
WOODRUFF, CHARLES E.	U. S. A.	86	Jeff.
WOODS, D. F.	1501 Spruce St., Phila.	64	U. of P.
WOODVILLE, J. L.	Sweet Springs, Monroe Co., Va.	44	U. of P.
YENNEY, ROBERT E.		93	
YOUNG, JAMES K.	222 S. 16th St., Phila.	84	U. of P.
YOUNG, W. W, Jr.		94	
ZACHERLE, O. F.	1301 N. 29th St., Phila.	87	Jeff.
ZIMMER, JOHN.	46 Rhine St., Rochester, N. Y.	93	U. of P.

CHRONOLOGICAL LIST OF THE MEMBERS

OF THE

Medical Boards of the Philadelphia Hospital

FROM 1768 to 1895.

By CHARLES K. MILLS, M.D.

[This list probably contains many omissions and not a few mistakes, as many difficulties have attended its preparation, in which have been consulted Dr. Agnew's "Medical History of the Philadelphia Almshouse," Thatcher's "Medical Biography," Rnschenberger's "History of the College of Physicians," the Catalogues of the Alumni of the Medical Department of the University of Penusylvania and of the Jefferson Medical College, the Annual Statements of the Boards of Guardians of the Poor and of the Board of Charities and Correction, and the written minutes of the Governing Boards since 1859. Members of the Medical Board have also been personally consulted in efforts at verification. I shall be glad to receive any corrections or additions from any one who may examine the table.—C. K. M.]

MEDICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
Cadwalader Evans	1768		
Thomas Bond	1768	1779	
Adam Kuhn	1774	1776	
Benjamin Rush	1774	1777	
Samuel Duffield	1774	1801	
Girardus Clarkson	1774	1777	
Girardus Clarkson	1788	1790	
Thomas Parke	1774	1779	
George Glentworth	1779	1781	
D. Jackson	1779	1781	
James Hutchison	1780	1781	Out-door physician.
—— Wilson	1780	1781	Out-door physician.
Caspar Wistar	1788	1790	
J. R. Rodgers	1788	1789	
Michael Leib	1788	1790	
John Morris	1788	1789	
Samuel P. Griffits	1788	1789	
N. B. Waters	1789	1790	
William Shippen	1789	1790	
—— Cumming	1795		

MEDICAL STAFF.—*Continued.*

Name.	Service began.	Service ended.	Remarks.
—— Pleasants		1797	Date of appointment not known.
Samuel Clements, Jr.	1796	1797	
William Boyce	1796	1801	
Samuel Cooper	1796	1796	
John Church	1797	1805	
Thomas C. James	1797	1811	Transferred to Obstetrical Staff.
John Proudfit	1801	1804	
Philip S. Physick	1801	1805	
Charles Caldwell	1801	1804	
Elijah Griffiths	1801	1810	
Benjamin L. Barton	1804	1805	
Samuel Stewart	1804	1810	
John Rush	1804		Elected, but declined to serve. Dr. Agnew mentions a Dr. Rush as resigning in 1821.
James Reynolds	1804	1807	
James Hutchinson	1805	1805	Served three months.
Isaac Cathrall	1805	1811	Transferred to Surgical Staff.
Peter Muller	1805	1811	Transferred to Surgical Staff.
John Syng Dorsey	1805	1811	
John Syng Dorsey	1814	1815	
Nathaniel Chapman	1807	1815	
Nathaniel Chapman	1822	1832	
Joseph Parrish	1807	1811	Transferred to Surgical Staff.
Samuel Stewart	1810	1822	
Joseph Klapp	1810		
Joseph Klapp	1815	1822	
Thomas Hewson	1811	1822	
Joseph Hartshorne	1818	1820	
Samuel Calhoun	1821	1822	
William P. C. Barton	1821	1822	
William E. Horner	1822	1832	Transferred to Surgical Staff.
Samuel Jackson	1822	1845	
John K. Mitchell	1822	1827	
Richard Harlan	1822	1822	Transferred to Surgical Staff.
Hugh L. Hodge	1822	1835	
Samuel George Morton	1827	1835	
Jacob Randolph	1832	1837	
William H. Gerhard	1835	1845	
Joseph Pancoast	1835	1837	Transferred to Surgical Staff.
William Ashmead	1835	1838	
William Ashmead	1841	1845	
N. Stuardson	1837	1838	
Robley Dunglison	1838	1845	
Edward Peace	1838	1841	
Meredith Clymer	1843	1845	

SURGICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
J. Cathrall...	1811	1822	Transferred from Medical Staff.
Peter Miller..	1811	1822	Transferred from Medical Staff.
Joseph Parrish	1811	1821	Transferred from Medical Staff.
John Rhea Barton	1820	1822	
William Gibson	1821	1822	
J. V. O. Lawrence	1822	1822	
Richard Harlan	1822	1838	Transferred from Medical Staff.
William E. Horner	1832	1835	Transferred from Medical Staff.
Joseph Pancoast	1837	1845	Transferred from Medical Staff.
Charles Bell Gibson	1838	1840	

OBSTETRICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
Thomas C. James.	1811	1821	Transferred from Medical Staff.
John Moore	1818	1821	
Henry Neill	1821	1835	
Nathan Shoemaker	1821	1827	
Charles Lukens	1827	1827	
B. Ellis	1827	1831	
F. S. Beattie	1831	1837	
Charles Wistar Pennock	1835	1845	
William D. Brinkle	1837	1839	
Charles Bell Gibson	1838	1840	
Robert M. Huston	1839	1845	
James McClintock	1840	1841	
William H. Gillingham	1841	1845	

Administration under Chief Resident Officer.

PHYSICIAN-IN-CHIEF.

Name.	Service began.	Service ended.	Remarks.
H. S. Patterson	1845	1845	

CONSULTANTS.

Name.	Service began.	Service ended.	Remarks.
William Byrd Page	1845		Consulting surgeon.
Meredith Clymer	1845		Consulting physician.
N. D. Benedict	1845	1845	Consulting accoucheur. Office abolished November 9, 1845.

PHYSICIANS-IN-CHIEF.

Name.	Service began.	Service ended.	Remarks.
N. D. Benedict	1845	1850	
—— Haines	1850	1853	
J. D. Stewart	1853	1854	
R. T. Coleman	1854	1854	
Archibald B. Campbell	1854	1854	

Administration by Residents-in-Chief and Board of Clinical Lecturers.

RESIDENTS-IN-CHIEF.

Name.	Service began.	Service ended.	Remarks.
Archibald B. Campbell	1854	1855	
Robert K. Smith	1855	1856	
Archibald B. Campbell	1856	1857	
James McClintock	1857	1858	All the visiting physicians resigned soon after the election of Dr. McClintock, as clinical instruction was abandoned.
Robert K. Smith	1858	1859	

LECTURERS ON CLINICAL MEDICINE.

Name.	Service began.	service ended.	Remarks.
J. L. Ludlow	1854	1857	
Robert Coleman	1854	1854	Did not accept election.
Gaspar Morris	1854	1855	Transferred to Obstetrical Department.
Joseph Carson	1855	1857	
Joseph Carson	1858	1859	
J. B. Biddle	1855	1857	
J. B. Biddle	1858	1859	
J. Aitken Meigs	1858	1859	
Samuel Dickson	1858	1858	Declined on account of ill health.
J. M. DaCosta	1858	1859	

LECTURERS ON CLINICAL SURGERY.

Name.	Service began.	Service ended.	Remarks.
Henry H. Smith	1854	1857	
D. H. Agnew	1854	1857	
D. H. Agnew	1858	1859	
John Neill	1855	1857	
R. P. Thomas	1855	1857	
W. S. Halsey	1858	1859	
Richard J. Levis	1858	1859	

LECTURERS ON OBSTETRICS AND DISEASES OF WOMEN AND CHILDREN.

Name.	Service began.	Service ended.	Remarks.
R. A. F. Peurose	1854	1857	
R. A. F. Penrose	1858	1859	
Wilson Jewell	1855	1857	
Caspar Morris	1855	1857	Trausferred from Medical Dep'm't.
E. McClellan	1858	1859	

MEDICAL STAFF.[1]

Name.	Service began.	Service ended.	Remarks.
J. L. Ludlow	1859	1885	
William F. Maybury	1859	1861	
Charles P. Tutt	1859	1866	
Robert Sucket	1859	1859	
J. M. Da Costa	1859	1865	
O. A. Judson	1861	1863	
George J. Ziegler	1863	1867	Transferred from Surgical Staff.
Alfred Stillé	1865	1872	
J. S. DeBenneville	1866	1866	
Edward Rhoads	1866	1870	
William Pepper	1867	1884	
H. C. Wood	1870	1883	Transferred to Neurological Staff.
James Tyson	1872	1889	
James Tyson	1893		Still Serving.
John M. Keating	1875	1877	
John M. Keating	1878	1880	Transferred to Obstetrical Staff.
Edward T. Brueu	1875	1889	
James C. Wilson	1875	1889	
John Guitéras	1875	1880	
Rolaud G. Curtain	1880		Still serving.
S. J. McFerran	1880	1884	
J. T. Eskridge	1882		Elected, but did not serve.
W. G. McConnell	1882		Elected, but did uot serve.
Joseph F. Neff	1884	1887	
John H. Musser	1885		Still serving.
William Osler	1885	1889	
F. P. Henry	1888		Still serving.
J. M. Anders	1889		Still serving.
William E. Hughes	1889		Still serving.
S. Solis-Cohen	1889		Still serving.
Eugene L. Vansant	1889		Still serving.
F. A. Packard	1892		Still serving.
Judson Daland	1892		Still serving.
Samuel Wolfe	1892		Still serving.
Julius Salinger	1892		Still serving.

[1] At this time the administration by a visiting medical board was resumed.

SURGICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
Samuel D. Gross	1859	1865	
D. Hayes Agnew	1859	1865	
R. J. Levis	1859	1870	
R. J. Levis	1882		Elected, but did not serve.
Edward L. Duer	1862	1863	Transferred to Obstetrical Staff.
R. S. Kenderdine	1859	1865	
J. W. Lodge	1864	1868	
W. H. Pancoast	1865	1885	
F. F. Maury	1865	1878	Transferred from Obstetrical Staff.
John H. Brinton	1866	1882	
Harrison Allen	1870	1878	
Samuel W. Gross	1874	1882	
N. L. Hatfield	1875	1884	
J. William White	1875	1889	
J. William White	1892		Still serving.
William G. Porter	1875		Still serving.
A. A. McDonald	1878	1881	
W. S. Janney	1877	1890	
George McClellan	1880	1890	
A. S. Roberts	1881	1887	
W. Joseph Hearn	1882		Still serving.
C. H. Thomas	1882	1884	
A. W. Ransley	1885	1892	
Lewis W. Steinbach	1885		Still serving.
John Blair Deaver	1887		Still serving.
Edward Martin	1888	1889	
Edward Martin	1892		Still serving.
Orville Horwitz	1889		Still serving.
Ernest Laplace	1889		Still serving.
James M. Barton	1890		Still serving.
J. Chalmers Da Costa	1895		Still serving.
Alfred C. Wood	1895		Still serving.

OBSTETRICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
R. A. F. Penrose	1859	1867	
John Wiltbank	1859	1859	
William D. Stroud	1859	1863	
Lewis Harlow	1859	1862	
George J. Ziegler	1859	1863	Transferred to Medical Staff.
A. H. Smith	1862	1864	
E. Schofield	1863	1870	
F. F. Maury	1864	1865	Transferred to Surgical Staff.
Edward L. Duer	1863	1883	Transferred from Surgical Staff.

OBSTETRICAL STAFF—*Continued.*

Name.	Service began.	Service ended.	Remarks.
R. M. Girvin	1865	1876	
J. S. Parry	1867	1876	
George Pepper	1870	1872	
J. V. Ingham	1872	1874	
W. A. Warder	1874	1881	
J. R. Burden, Jr.	1874	1876	
E. E. Montgomery	1877	1884	
E. E. Montgomery	1886		
James B. Walker	1876	1880	Transferred to Medical Staff.
S. S. Stryker	1876	1889	
G. W. Linn	1876	1882	
M. B. Musser	1877	1887	
W. H. Parish	1876	1889	
John M. Keating	1880	1890	Transferred from Medical Staff.
Clara Marshall	1882	1895	
E. P. Bernardy	1882	1884	
Hannah P. Croasdale	1882		Elected, but did not serve.
Theophilus Parvin	1884	1892	
Donnell Hughes	1884	1884	
Eliott Richardson	1886	1886	
Barton C. Hirst	1887		Still serving.
Edward P. Davis	1889		Still serving.
William Easterly Ashton	1889		Still serving.
Robert H. Hamill	1890		Still serving.
George I. McKelway	1890		Still serving.
J. W. West	1892	1894	
R. C. Norris	1894		Still serving.
J. M. Fisher	1894		Still serving.
W. Frank Haehnlen	1895		Still serving.
Elizabeth L. Peck	1895		Still serving.

NEUROLOGICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
Charles K. Mills	1877		Still serving.
H. C. Wood	1883	1887	
H. C. Wood	1887	1888	
Robert Bartholow	1887	1888	
Francis X. Dercum	1887		Still serving.
James Hendrie Lloyd	1888	1889	
James Hendrie Lloyd	1890		Still serving.
Wharton Sinkler	1888		Still serving.
C. H. Bradfute	1889	1890	

OPHTHALMOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
E. O. Shakespeare	1877	1889	
G. E. de Schweinitz	1887		Still serving.
Charles H. Thomas	1888	1888	Acting ophthalmologist.
George M. Gould	1889	1894	
Charles A. Oliver	1894		Still serving.

DERMATOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
F. F. Maury	1870	1870	Acting dermatologist.
Louis Duhring	1870	1877	
Louis Duhring	1877	1889	Acting dermatologist.
Henry W. Stelwagon	1887		Still serving.
J. A. Cantrell	1889		Still serving.

LARYNGOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
C. Jay Seltzer	1890		Still serving.
George M. Marshall	1890		Still serving.

PATHOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
James Tyson	1871	1875	
R. M. Bertolet	1872		
Joseph Berens	1876	1879	
E. O. Shakespeare	1882	1889	
Henry F. Formad	1887	1892	
John Guitéras	1892		Still serving.
W. M. L. Coplin	1892	1895	
E. B. Sangree	1895		Still serving.

BACTERIOLOGIST.

Name.	Service began.	Service ended.	Remarks.
E. O. Shakespeare	1889	1894	
A. Ghriskey	1894		

ASSISTANT PATHOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
L. L. Hatch	1889	1891	
H. W. Cattell	1889		Still serving.
William B. Jameson	1892		Still serving.
Ernest B. Sangree	1892	1895	
David Bevan	1892	1895	

CURATORS.

Name.	Service began.	Service ended.	Remarks.
D. Hayes Agnew	1860	1867	
William Pepper	1867	1871	
R. M. Bertolet	1871	1872	
R. M. Bertolet	1875	1876	
James Tyson	1872	1875	
Joseph Berens	1876	1879	
E. O. Shakespeare	1880	1882	

MICROSCOPISTS.

Name.	Service began.	Service ended.	Remarks.
James Tyson	1866	1872	
R. M. Bertolet	1872	1875	
Thomas B. Reed	1875	1876	
H. F. Formad	1880	1892	

PHYSICIANS TO THE INSANE DEPARTMENT.

Name.	Service began.	Service ended.	Remarks.
L. Henley	1849	1852	Assistant physician in charge.
J. H. Benton	1852	1852	Assistant physician in charge.
L. Henley	1852	1854	From 1854 to 1859 no one regularly in charge.
Samuel W. Butler	1859	1866	
D. D. Richardson	1866	1880	
D. D. Richardson	1881	1885	
A. A. McDonald	1880	1881	
Philip Leidy	1885	1887	
William H. Wallace	1887	1887	
George M. Wells	1887	1890	
Daniel E. Hughes	1890		Still serving.

CONSULTING PHYSICIAN TO THE INSANE DEPARTMENT.

Name.	Service began.	Service ended.	Remarks.
S. Weir Mitchell	1884	1886	
Horatio C. Wood	1884	1885	
Charles K. Mills	1884	1887	
Charles K. Mills	1890		Still serving.
Andrew Nebinger	1885	1886	
James A. Simpson	1886	1887	
Philip Leidy	1886	1887	
F. X. Dercum	1890		Still serving.
Wharton Sinkler	1890		Still serving.
James Hendrie Lloyd	1890		Still serving.

Registrars.

MEDICAL.

Name.	Service began.	Service ended	Remarks.
W. A. Edwards	1885	1886	
C. J. Seltzer	1886	1890	
F. A. Paekard	1890	1892	
Alfred Stengel	1892		Still serving.
H. Toulmin	1895		Still serving.

SURGICAL.

Name.	Service began.	Service ended.	Remarks.
Edward Martin	1885	1888	Transferred to Surgieal Staff.
C. B. Penrose	1888	1892	
J. C. Da Costa	1892	1895	Transferred to Surgical Staff.

OBSTETRICAL.

Name.	Service began.	Service ended.	Remarks.
H. A. Pardee	1885	1887	
Edward P. Davis	1887	1888	
R. H. Hamill	1888	1890	
R. C. Norris	1890	1894	Transferred to Obstetrieal Staff.

NERVOUS.

Name.	Service began.	Service ended.	Remarks.
Guy Hinsdale	1885	1892	
Augustus A. Eshner	1891		Still serving.

LIST OF MEMBERS OF THE MEDICAL BOARD.

With Addresses, Place and Time of Graduation, Date of Appointment to the Philadelphia Hospital, and Positions held in other Institutions.

In the main this list represents the order of seniority of the different members of the medical board; but in a few instances it does not, as some of the present members are serving for a second period. Some also have been elected during the same year, or even at the same meeting of the governing board, and practically the latter do not differ in seniority.

James Tyson, M.D., 1506 Spruce street. Graduate of Univ. Penna., 1863. Appointed 1872; served until 1889; re-appointed 1893. Professor of Clinical Medicine in the University of Pennsylvania, and Physician to the University Hospital.

J. William White, M.D., 1810 South Rittenhouse Square. Graduate of Univ. Penna., 1871. Appointed in 1875; served until 1889; re-appointed in 1892. Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University and to the German Hospitals.

Charles K. Mills, M.D., 1909 Chestnut street. Graduate of Univ. Penna., 1869. Appointed 1877. Professor of Mental Diseases and of Medical Jurisprudence in the University of Pennsylvania, and Dean of the Faculty of the Auxiliary Department of Medicine in the University; Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic; Clinical Professor of Nervous Diseases in the Woman's Medical College of Pennsylvania; Consulting Physician to St. Clement's Hospital for Epileptics; Consultant to St. Joseph's Hospital, to the West Philadelphia Hospital for Women, to the New Jersey Training-School for Feeble-Minded Children, and to the Training-School for Feeble-Minded Children at Elwyn.

Roland G. Curtin, M.D., 22 South Eighteenth street. Graduate of Univ. Penna., 1866. Appointed 1880. Consulting Physician to the Rush Hospital for Consumptives, to St. Timothy's Hospital, and to the Midnight Mission; Visiting Physician to the Presbyterian Hospital; Lecturer on Physical Diagnosis in the University of Pennsylvania; Assistant Physician to the Hospital of the University of Pennsylvania; President of the American Climatological Society.

W. Joseph Hearn, M.D., 1130 Walnut street. Graduate of Jefferson Medical College, 1867. Appointed 1882. Visiting Surgeon to the Jefferson Medical College Hospital; Clinical Professor of Surgery in the Jefferson Medical College.

Lewis W. Steinbach, M.D., 1309 North Broad street. Graduate of Jefferson Medical College, 1880. Appointed 1885 Surgeon to the Jewish Hospital; Professor of Surgery in the Philadelphia Polyclinic.

JOHN H MUSSER, M.D., 1927 Chestnut street. Graduate of Univ. Penna., 1877. Appointed 1885. Assistant Professor of Clinical Medicine in University of Pennsylvania; Physician to the Presbyterian Hospital; Consulting Physician to the Woman's Hospital and to the West Philadelphia Hospital for Women; President of the Pathological Society of Philadelphia.

HENRY W. STELWAGON, M.D., 223 South Seventeenth street. Graduate of Univ. Penna., 1875. Appointed 1887. Clinical Professor of Dermatology in the Jefferson Medical College, and in the Woman's Medical College; Physician to the Skin Department of the Northern Dispensary and of the Howard Hospital.

FRANCIS X. DERCUM, M.D., 810 North Broad street. Graduate of Univ. Penna., 1877. Appointed 1887. Clinical Professor of Diseases of the Nervous System in the Jefferson Medical College; Visiting Physician to the St. Clement's Hospital for Epileptics; Consulting Neurologist to St. Agnes' Hospital; Assistant Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases.

G. E. DE SCHWEINITZ, M.D., 1401 Locust street. Graduate of Univ. Penna., 1881. Appointed 1887. Professor of Ophthalmology in Philadelphia Polyclinic and School for Graduates; Clinical Professor of Ophthalmology in Jefferson Medical College; Ophthalmic Surgeon to the Children's Hospital, to the Orthopedic Hospital and Infirmary for Nervous Diseases and to the Methodist Hospital; Ophthalmologist to the Church Home, Angora, and to the Home of the Merciful Saviour for Crippled Children; Consulting Ophthalmologist to the Chester County Hospital, to the Bryn Mawr Hospital and to the Hospital for Epileptics and Dispensary of St. Clement's Church.

JOHN B. DEAVER, M.D., 1634 Walnut street. Graduate of Univ. Penna., 1878. Appointed 1887. Assistant Professor of Applied Anatomy in the University of Pennsylvania; Attending Surgeon to the German Hospital; Consulting Surgeon to St. Agnes', Germantown, St. Timothy's Hospital, to the Home for Feeble-Minded Children, Elwyn, and to the Southern Home for Destitute Children.

BARTON COOKE HIRST, M.D., 1821 Spruce street. Graduate of Univ. Penna., 1883. Appointed 1887. Professor of Obstetrics in the University of Pennsylvania; Obstetrician to the University and Maternity Hospitals; Gynecologist to the Orthopedic Hospital.

FREDERICK P. HENRY, M.D., 1635 Locust street. Graduate of College of Physicians and Surgeons, New York, 1868. Appointed 1887. Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania; Physician to the Home for Consumptives at Chestnut Hill.

EDWARD MARTIN, M.D., 415 South Fifteenth street. Graduate of Univ. Penna., 1883. Appointed 1888; served until 1889; re-appointed 1892. Served as Surgical Registrar from 1885 to 1888. Clinical Professor of Genito-Urinary Diseases in the University of Pennsylvania.

JAMES HENDRIE LLOYD, M.D., 3910 Walnut street. Graduate of Univ. Penna., 1878. Appointed 1888; served until December, 1889; reappointed, 1890. Physician to the Methodist Episcopal Hospital; Physician to the Home for Crippled Children; Neurologist to the Pennsylvania Training School for Feeble-Minded Children at Elwyn; Consulting Neurologist to the State Hospital for the Chronic Insane at Wernersville.

EDWARD P. DAVIS, M.D., 250 South Twenty-first street. Graduate of Rush Medical College, Chicago, 1882. Appointed 1888. Professor of Obstetrics and Diseases of Infancy in the Philadelphia Polyclinic; Clinical Professor of Obstetrics in the Jefferson Medical College.

WHARTON SINKLER, M.D., 1606 Walnut street. Graduate of Univ. Penna., 1868. Appointed 1888. Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases; Consulting Physician to the Pennsylvania Hospital for Epilepties; Neurologist to the Bryn Mawr Hospital; Consulting Neurologist to the Pennsylvania State Hospital for the Chronic Insane.

J. M. ANDERS, M.D., Ph.D., 1605 Walnut street. Graduate of Univ. Penna., 1877. Appointed 1889. Professor of the Principles and the Practice of Medicine and of Clinical Medicine in the Medico-Chirurgical College; Physician to the Medico-Chirurgical and Samaritan Hospitals.

W. E. HUGHES, M.D., 3726 Baring street. Professor of Clinical Medicine in the Medico-Chirurgical College; Visiting Physician to the Medico-Chirurgical Hospital; Pathologist to the Presbyterian Hospital.

SOLOMON SOLIS-COHEN, M.D., 219 South Seventeenth street. Graduate of Jefferson Medical College, 1883. Appointed 1889. Professor of Clinical Medicine and Applied Therapeutics in the Philadelphia Polyclinie; Lecturer on Clinical Medicine in the Jefferson Medical College; Consulting Physician to the Jewish Hospital; Physician to the Rush Hospital; Consulting Laryngologist to the Pennsylvania Institution for Feeble-Minded Children.

EUGENE L. VANSANT, M.D., 1929 Chestnut street. Graduate of Jefferson Medieal College, 1884. Appointed 1889. Lecturer on Clinical Medieine, Jefferson Medical College; Surgeon to the Throat, Nose and Ear Department of the Howard Hospital.

ORVILLE HORWITZ, M.D., 1115 Walnut street. Graduate of Jefferson Medical College, 1883. Appointed 1889. Clinical Professor of Genito-Urinary Diseases, Jefferson Medical College; Professor of Genito-Urinary Surgery, Philadelphia Polyclinic.

ERNEST LAPLACE, M.D., 1617 Arch street. Graduate of Univ. La., 1884. Appointed 1889. Professor of Clinical Surgery in the Medico-Chirurgical College.

WILLIAM EASTERLY ASHTON, M.D., 2011 Walnut street. Graduate of Univ. Penna., 1881; Graduate of Jefferson Medical College, 1884. Appointed 1889. Professor of Gynecology in the Medico-Chirurgical College; Gynecologist to the Medico-Chirurgical Hospital,

J. ABBOTT CANTRELL, M.D., 315 South Eighteenth street. Graduate of Jefferson Medical College, 1885. Appointed 1889. Professor of Diseases of the Skin in the Philadelphia Polyclinic and College for Graduates in Medicine; Dermatologist to the Philadelphia and Frederick Douglass Memorial Hospitals, and to the Southern Dispensary.

HENRY W. CATTELL, M.D., 3455 Woodland avenue. Gradnate of Univ. Penna., 1887. Appointed 1889. Demonstrator of Morbid Anatomy in the University of Pennsylvania; Pathologist to the Presbyterian Hospital and to the Institution for Feeble-Minded Children, at Elwyn; Presector to the American Anthropometric Society.

W. B. JAMESON, M.D., 767 North Fortieth street. Graduate of Univ. Penna., 1886. Appointed 1890.

JAMES M. BARTON, M.D., 1337 Spruce street. Graduate of Jefferson Medical College, 1868. Appointed 1890. Surgeon to the Jefferson Medical College Hospital.

C. JAY SELTZER, M.D., 1501 Walnut street. Graduate of Univ. Penna., 1881. Appointed 1890.

GEORGE MORLEY MARSHALL, M.D., 1819 Spruce street. Graduate of Univ. Penna., 1886. Appointed 1890. Attending Physician to St. Joseph's Hospital and Chief of its Throat Dispensary.

ROBERT H. HAMILL, M.D., 330 South Sixteenth street. Graduate of Univ. Penna., 1878. Appointed 1890. Obstetrician to Maternity Hospital; Gynecologist to the Howard Hospital.

GEORGE I. MCKELWAY, M.D., 1612 Locust street. Graduate of Univ. Penna., 1889. Appointed 1890.

FREDERICK A. PACKARD, M.D., 110 South Eighteenth street. Graduate of Univ. Penna., 1885. Appointed Medical Registrar, 1890; served until 1892; appointed on the Medical Staff, 1892. Instructor in Physical Diagnosis, University of Pennsylvania; Visiting Physician to the Children's Hospital, and to the Out-Patient Department of the Pennsylvania Hospital.

RICHARD C. NORRIS, M.D., 500 North Twentieth street. Graduate of Univ. Penna., 1887. Appointed Registrar, 1890; Obstetrical Staff, 1894; Lecturer on Clinical and Operative Obstetrics, University of Pennsylvania; Physician in Charge, Preston Retreat; Gynecologist to Methodist Episcopal Hospital; Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital for Women and Children.

J. CHALMERS DA COSTA, M.D., 2050 Locust street. Graduate of Jefferson Medical College, 1885. Appointed registrar, 1890. Appointed on Surgical Staff, 1895. Demonstrator of Surgery, Jefferson Medical College; Chief Assistant Surgeon, Jefferson Medical College Hospital.

SAMUEL WOLFE, A.M., M.D,, 1701 Diamond street. Graduate of Univ. Penna., 1873. Appointed 1892. Formerly Clinical Professor of Nervous Diseases, Medico-Chirurgical College; formerly Professor of Physiology, Medico-Chirurgical College; Neurologist to Medico-Chirurgical and Samaritan Hospitals.

JULIUS L. SALINGER, M.D., 1510 North Eighth street. Graduate of Jefferson Medical College, 1886. Appointed 1892. Lecturer on Renal Diseases, Jefferson Medical College; Chief of the Medical Clinic, Jefferson Medical College Hospital.

JOHN GUITÉRAS, M.D., 3914 Sansom street. Graduate of Univ. Penna., 1873. Appointed 1892. Served on the Medical Staff from 1875 to 1880. Professor of Pathology in the University of Pennsylvania; Pathologist to the Hospital of the University of Pennsylvania; President of the Section of Pathology in the Pan-American Congress.

AUGUSTUS A. ESHNER, M.D., 224 South Sixteenth street. Graduate of Jefferson Medical College, 1888. Appointed 1891. Professor of Clinical Medicine in the Philadelphia Polyclinic.

ERNEST B. SANGREE, A.M., M.D., 2020 Arch street. Graduate of Medico-Chirurgical College of Philadelphia. Appointed 1892. Assistant Professor of Pathology in Medico-Chirurgical College; Demonstrator of Histology in Phila. Dental College.

ALFRED STENGEL, M.D., 332 South Seventeenth street. Graduate of Univ. Penna., 1889. Appointed 1892. Pathologist to German Hospital; Instructor in Clinical Medicine, University of Pennsylvania; Assistant Physician to the University Hospital; Physician to Howard Hospital.

JOHN M. FISHER, M.D., 1527 Wallace street. Graduate of Jefferson Medical College, 1884. Appointed 1894. Chief of the Department of Diseases of Women and Demonstrator of Gynecology in the Jefferson Medical College Hospital.

THOMAS G. ASHTON, M.D., 1533 Pine street. Graduate of Jefferson Medical College, 1888. Appointed 1894. Chief of the Out-Patient Medical Department of the Jefferson Medical College Hospital; Demonstrator of Clinical Medicine, Jefferson Medical College; Visiting Physician to St. Mary's Hospital.

ALBERT GHRISKEY, M.D., 3916 Walnut street. Graduate of Univ. Penna., 1880. Appointed 1894.

CHARLES A. OLIVER, M.D., 1507 Locust street. Graduate of Univ. Penna., 1876. Appointed 1894. Attending Surgeon to Wills' Eye Hospital; Ophthalmic Surgeon to Presbyterian Hospital; Consulting Ophthalmic Surgeon to St. Agnes', St. Mary's, St. Timothy's and Maternity Hospitals; Consulting Ophthalmologist to State Hospital for Chronic Insane of Penna., and to State Hospital for the Insane for the S. E. District of Penna.; Ophthalmologist to the Friends' Asylum for the Insane, Frankford.

H. TOULMIN, M.D., 4009 Chestnut street. Graduate of Univ. Penna., 1889. Appointed 1895. Instructor in Physical Diagnosis, Univ. of Penna. Visiting Physician, Phila, Home for Incurables.

W. FRANK HAEHNLEN, M.D., 1616 Walnut street. Graduate of Univ. Penna., 1882. Appointed 1895. Professor of Obstetrics, Medico-Chirurgical College; Obstetrician to Medico-Chirurgical and Maternity Hospitals; Medical Director and Gynecologist, Samaritan Hospital.

ALFRED C. WOOD, M.D., 214 South Fifteenth street. Graduate of Univ. Penna., 1888. Appointed 1895. Assistant Surgeon to the University Hospital; Instructor in Clinical Surgery in the University of Pennsylvania.

ELIZABETH L. PECK, M.D., 819 North Fortieth street. Graduate Woman's Medical College of Penna., 1885. Appointed 1895. Visiting Physican, West Phila. Hospital for Woman; Instructor in Materia Medica and Therapeutics, Woman's Medical College of Penna.

OFFICIAL CHANGES.

WHEN the second volume of the PHILADELPHIA HOSPITAL REPORTS was printed in 1893, the Department of Charities and Corrections was constituted of the following Directors:

William H. Lambert, President; William D. Gardner, Treasurer; Alfred Moore, John Huggard and James W. Walk, M.D. The members were assigned to duty as follows:

Bureau of Charities, Mr. Moore, Chairman, Mr. Gardner; Bureau of Correction, Mr. Huggard, Chairman, Dr. Walk, President Lambert being *ex-officio* a member of both Bureaus.

The illness of Mr. Huggard, which during the greater part of the year 1893 had prevented his active co-operation in the affairs of the Department, having ended in his death on the 24th of January, 1894, a special meeting of the Board was held on the next day to take action upon the sad event. The following minute, prepared by Dr. Walk and Mr. Moore, was adopted:

"The members of this Board have learned with deep sorrow of the death of our friend and colleague, John Huggard, and desire to place upon record our appreciation of his character as a man, a citizen and a public officer.

"Always actuated by a high sense of civic duty and a sincere sympathy with the unfortunate, he brought to the service of this department qualities which peculiarly fitted him for his duties as a director—a sound judgment, a ripe experience, and a kindness of heart which ever sought to temper justice with mercy.

"A man of unquestioned integrity and exalted patriotism, his death leaves a void in many hearts, and his memory will be cherished as that of a good citizen, a faithful public officer and a true friend."

Mr. John Shallcross was appointed by Mayor Stuart to fill the vacancy caused by the lamented death of Mr. Huggard, and was assigned to the Bureau of Correction, of which Dr. Walk was made Chairman.

On October 1, 1895, a change was made by the assignment of Mr. Gardner to the Bureau of Correction, of which Mr. Shallcross was made Chairman, and Dr. Walk to the Bureau of Charities.

The chief officials of the department remain the same.

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